

GERIATRIC MEDICINE

The Care of the Aging and the Aged

Edited by

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THE LONGER MEN LIVE
THE MORE TIME THERE IS TO THINK
TO THINK IS TO GROW
AND GROWING LIVE

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PREFACE TO THE SECOND EDITION

THE limitations of geriatric medicine have loomed large and discouraged many. The false assumption that it was concerned only with the senile has led to an attitude of futility. Though clinical geriatric medicine does treat of the aged, it is even more concerned with the aging, and with the prevention of premature senile infirmity. Too often the benefits of modern medicine have been denied the elderly on the fatuous argument that the patient is too old to live long anyway. Old people tolerate surgery and severe illness surprisingly well if one but applies intelligent effort on their behalf. Proper preoperative attention to nutrition and circulatory competence lowers surgical mortality dramatically. Social attitudes toward the aged have increased the inertia. True it is that the life span is limited, but it is not necessary that the evening of life be clouded by prolonged invalidism or chilled by parasitic uselessness. Life should have depth and breadth as well as length. Lowered homeostatic efficiency and accumulated injuries limit the possible accomplishments of clinical medicine for the aged, but do not preclude them. Therapy is rarely dramatically curative. Control and retardation of progressive deterioration, however, can accomplish much that is worth while.

The potentialities of geriatric medicine are what we make them. They have been, as yet, hardly explored, let alone developed. There is still much to be learned, especially concerning the fundamentals of the biology of senescence and the complex etiology of the degenerative diseases so frequent in later maturity. The development and maturation of knowledge are as asym-

then all progress will have ceased. Awareness of imperfections is requisite to improvement just as awareness of ignorance is the prelude to learning.

That more extensive application of what is already known will lead to gratifying results is our earnest conviction, based upon personal experience. It is this conviction which justifies this work. The rewards of geriatric medicine are great, though perhaps less dramatic than those of other fields of practice. There is an intense and lasting satisfaction in solving obscure, complicated diagnostic problems, in correlating subtle psychic and physiologic mechanisms and processes, and in applying prophylactic and constructive

measures to maintain and health than treatment of the sequelae of health.

The social values of geriatric medicine have been too little appreciated. The elderly constitute a heretofore unappreciated but precious fraction of our human resources. Wisdom and judgment are derived from experience. Time

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SECTION I

GENERAL CONSIDERATIONS

CHAPTER I

FOUNDATIONS OF GERIATRIC MEDICINE

EDWARD J. STIEGLITZ

ORIENTATION

PERSPECTIVE, or analysis of the relation of parts to one another and to the whole, is essential to comprehension of any concept, problem, situation, or object. No problem is an isolated entity. To be fully understood it must be viewed and studied in relation to other issues and subjects. Perspective as here applied includes not only space and time relationships, but also analysis of the composition, activities, and intimate interactions of all component elements. To fully understand man is the aim of every true physician. Man is infinitely complex and *one of his fundamental attributes is that he ages and as he ages, changes*.

Full comprehension of any problem demands the application of study from three distinctive viewpoints to obtain the necessary perspective. Omission of any one of these inevitably results in incomplete, distorted, false, and asymmetric understanding. First we need to study the problem as a unit with the naked eye, secondly we must take it apart and apply magnification to its component parts and activities, and lastly it is necessary to step back far enough so that it may be inspected and investigated telescopically, and thus related to its external environment. In other words, a close-up view without a background invariably results in a false set of values. Likewise, long range perspective without detailed magnification of the component elements and multiple facets of a problem yields an equally distorted view.

Translated into other terms, full comprehension of the potentialities of medicine involves three approaches. (1) the indivisible individual, psyche and soma as one, is the unit of study of clinical medicine. (2) magnification of the component parts—the biochemical reactions, electrical potentials, and cellular units which make up the individual, is the concern of the biological sciences upon which clinical medicine depends for understanding the entity, man, and (3) man as a member of society, a unit of the external environment, is the concern of social medicine. He is affected by other people, climate, housing, food, threats of disaster, and at the same time his behavior affects his environment.¹ With aging individuals these relations are of particular significance. Geriatric medicine, if it is to develop its full and significant potentialities, must maintain this triad of perspectives.

Looking further afield than our own small selves is difficult and discomfiting. Thus we try to avoid it. Nevertheless, study beyond the narrow confines of specialized technics is necessary for the maintenance of perspective. The specialist has been defined as the man who knows more and more about less and less until ultimately he knows everything about nothing. There is a strong urge to stay confined within the narrow rut of routine, it seems so secure. But the security is only apparent and hardly worth the inevitable price of persistent deepening of the rut until it becomes indistinguishable from an intellectual grave.

Aging is a part of living. All living matter ages and as it ages, changes. Aging involves every one of the innumerable aspects of life. It begins with conception and ends only with death. Thus growth, development, and maturation are just as much consequences of the occult processes of aging as are the atrophies and degenerations of senility. Evolution and involution are both affected by aging, pediatrics and geriatrics are closer than many realize. Gerontology is the science of aging in its broadest sense. The aged and aging are not the same, the aged are people, aging is a process. Whereas pediatrics is concerned with the early stages of the life span, geriatrics is that branch of medical science concerned with the physiologic and pathologic problems of older individuals. Geriatrics is thus but a part, a subdivision, of the broader field of gerontology.

Gerontology involves all of the many varied divisions of the biological, physical, and social sciences. Nonliving matter ages also, witness the changes occurring in metals and in colloids with the passage of time. Gerontology is concerned with aging as a biologic phenomenon as well as with the consequences of age changes. Aging must be considered not only from the point of view of an individual but also from the point of view of the species, for various species are unquestionably of different evolutionary, or involutional, age. Cultural maturation of social groups is a phenomenon of aging.

These preliminary comments make it quickly obvious that gerontology is so broad a field and involves so many aspects of science that it is necessary to have some skeletal and basic plan in order to make logical correlation of the problems. A simple and pragmatic division into three major categories clarifies the relationships.

THE PROBLEMS OF GERONTOLOGY

- 1 The Biology of Aging
 - 1 1 The phenomena of evolution or development
 - 1 2 The phenomena of involution or senescence
- 2 The Clinical Problems of Aging Man
 - 2 1 Pediatric medicine
 - 2 2 Geriatric medicine
 - 2 21 Normal senescence and senility
 - 2 211 Physical aspects
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 - 2 22 Diseases of the Senescent Period
 - 2 221 Physical disorders
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- 3 The Socio-economic Problems of Aging Mankind

3 1 Economic problems

3 11 Unemployment in relation to age

3 12 Chronic illness in and after maturity

3 13 Mental diseases in senescents

3 2 Industrial problems of aging personnel

3 3 Cultural problems

3 31 Adult education

3 32 Family and social attitudes toward age

3 33 Intellectual and emotional maturity

*This classification of the problems of aging is highly condensed and attempts to present merely the major divisions or perspectives. Refinement of the classification would reveal almost innumerable ramifications of this immense field. Such a task is beyond the scope of the present volume, where our primary concern is with the clinical problems of individual aging men and women, the application of the second division of gerontology. Nevertheless, the triad of primary categories or perspectives of gerontology is intimately and inseparably related, both theoretically and pragmatically. Here theory and practice are not in conflict. Though widely differing disciplines and techniques of scientific research must be applied to study of the various fields, the observations and conclusions derived from such investigations will fit into the broad pattern and thus amplify the whole.*²

The first major division, the *biology of aging* as a process or series of processes, considers the cell as the basic unit, the second division, concerned with *clinical problems*, constitutes the organism of man as its unit, and finally, problems of aging are also presented by the *social organism* of mankind, which is the fundamental unit of the third category. Obviously, advances in knowledge in any one subdivision will depend upon preceding or simultaneous progress in the other fields. It cannot be overemphasized that the more that is known about the fundamental biologic mechanisms of the aging process, the more effectively can clinical medicine understand and treat the problems of the aging and the aged. Similarly, the greater the clinical knowledge concerning the changes, capacities, and limitations of normal persons as they age and the prevention, control, or retardation of the progressive disorders of later years, the more intelligent can be the attack upon the serious and complex social problems which are becoming increasingly urgent every day.

Geriatric medicine, or the medical care of the aging and the aged, must include all fields of medical practice applied to those past the prime of life. Knowledge in this field of medical science and practice is not so crystallized that one can be dogmatic. There is more to be learned than we already know. But application of what is already known lags far behind existing knowledge. Geriatric medicine, as all clinical medicine, is both a science and an art. Herein we have collected contributions from those eminent in their special fields of medicine, to guide and advise practicing physicians, general practitioners and specialists alike. Geriatric medicine itself is not a limited specialty in clinical practice, but rather the application of the special knowledge and understanding which comes with appreciation of the biologic, structural, physiologic, psychologic, and sociologic changes which come with aging. The scientific basis of geriatric medicine is the body of knowledge pertaining to the biology of senescence³ and the disorders characteristic of later maturity. But the practice of geriatric medicine operates in a social environment and the

external factors of that environment affect its application most emphatically. The three facets or perspectives of gerontology must be correlated.

The scope of geriatrics will be determined by whatever definition we apply to this field of medicine. To some, geriatrics means only the care of the senile infirm—a sterilizing and restricting definition which would reduce geriatric therapy to mere palliation. Geriatric medicine can be and should be much more than the care of the truly aged. It includes the problems of aging, the vastly intriguing and significant problem of how we become old. To lift a particularly virile phrase from slang: How do the old get that way? Geriatric medicine is that part of medical science and practice which pertains to the health of the *aging* and the *aged*. It thus includes not only the problems of disease in those past the prime of life but also the complex phenomena of *normal senescence*. To live is to age and aging is change. Aging may be divided into several obviously different periods: (1) evolution, (2) maturation, (3) senescence, and (4) senility. It is with the last two phases that geriatric medicine is concerned.⁴

BIOLOGY OF AGING

The study of aging as a process has been conspicuously neglected until very recently. Biologists and physicians have been strangely content to take the phenomena of aging as a matter of course. There has been a vague academic interest in these questions for a long time, but there seemed little urgency, and interest was scattered and isolated. For centuries the philosophers and poets seemed to be the only ones interested in the aged. The ancient Greeks held great reverence for their aged seers, but this was due in part to the fact that in those days the very old were objects of curiosity because of their rarity. Cicero wrote his magnificent *De Senectute* several years before his death at sixty three. He considered himself an old man in the fifties and justly so, for few of his contemporaries survived to such ripeness. A young world is interested in youth. Science is young, culture is young, and both have been concerned primarily with exploration of the physical world about man rather than with the study of man himself. Science normally attacks the simpler problems first, particularly if they are conspicuous. Growth and development are quick and dramatic, they excited scientific investigation before the other end of the life span seemed of much moment.

In our present stage of cultural development, aging has been represented
 violent days of physical pro-
 for the aged. Aging was con-
 r of death, and thus feared,
 shunned and often abhorred. Our culture still attempts to avoid the issue, takes pride in youth, and deplors aging. By lies and ill fitting cosmetic subterfuges men and women try to conceal the signs of their age. There is often a sense of shame in admitting advanced years and many are those who insist upon deluding themselves that they are young, though their devices for misleading others are pathetically transparent. This fear of senescence is largely fear of loss of social respect, for the ill repute of old age is transmitted from generation to generation. It is fostered by youth, which wishes to claim the world as its own.

The evil reputation of age arises from a tragic misconception. Assumption, superficial evidence, and the unthinking tendency to make sweeping

generalizations perpetuate the erroneous idea that senescence means only decline. This is not true. To compensate for the decrements in certain physiologic and mental capacities there occur increments in other capacities. With waning powers of speed in adaptation comes compensatory increase in skills. Failing memory for petty details is often offset by bettered judgment (See Chapter 5.) Broader vision and greater consciousness of social responsibilities are among the gains of later maturity. By training and preparation for senescence these assets may be developed, in some respects these gains exceed the losses in the balance sheet of life. The older and wiser culture of the Chinese considers aging as an opportunity for development, rich in potential growth of character and wisdom. Were our culture so mature, many of the problems of gerontology would be far nearer solution, for the apathy and inertia obstructing study into the problems of senescence would then be replaced by an enthusiastic desire to learn.

These depressing emotional elements are significant forces in retarding the development of an organized study of aging. The momentum of centuries is not quickly overcome. But a change in attitude will come and it must come soon, for there is now a true urgency in the need to know more about aging and the aged. Man is a utilitarian creature and even pure scientific research is in part motivated by practical necessity. The present situation of dramatically rapid increase in the number and proportion of the elderly in our population is wholly without precedent. The aged are here now, there will be many more of them in the years to come. Never before in the history of mankind has a like problem arisen, plagues, wars, and famines have many times seriously changed the balance of population structure, but never before in the direction of a preponderance of the elderly such as is occurring today.

The need to learn about aging is urgent.⁵ The apparent boon of great longevity may become a curse, a terrible danger. Long life without health is not only an individual, personal tragedy, but a social evil seriously threatening national economy. Increased longevity with health and useful vigor into senility may be made an incalculably valuable asset to the commonwealth, if the potentialities of the elderly are wisely developed, guided, and utilized. The future course of events, economic destruction or great enrichment of human life, will depend in a large measure upon the science and art of medicine.⁶ Such is the vast responsibility of geriatric medicine.

It is impossible in our present ignorance to say what aging is, what it does, or why and how it does it. Very little is known, much of what we think we know is merely suspicion. Some is more purely wishful thinking. But the consciousness of ignorance is distinctly encouraging.

Aging is continuous. It begins with the onset of life at conception, it can be terminated only with death. Aging may be defined, perhaps, as those changes which are introduced by the factor of time in living. As it involves all of life, it must be studied by the many disciplines of the biologic sciences in order to be understood. We may start at the beginning by attempting to answer four apparently simple questions. (1) What is aging? (2) What does it do? (3) Why is it? (4) What accelerates or retards it? Simple though these questions appear, their solution will be the result of a long and arduous exploration, involving many disappointments, many failures, wearisome following of trails which lead to cul de sacs, and the efforts of innumerable investigators.

Rate of Aging There are many kinds of aging and many kinds of time. Geologic time is but a moment compared to the periods of astronomic time. The life span of one individual is as but a second in the progress of mankind's ascent as a species. Even for a given individual time actually has no constant rate. Certainly psychologic time varies in its rate in direct proportion to the interest and excitement of the moment: a half hour's wait in a physician's or a dentist's reception room can seem eternal, whereas during courtship hours seem but brief moments. Furthermore, the rate of time's progress varies in the impressions of different individuals, for they vary in their rate of living.

Just as time varies, so does the rate of aging. Most important is the recognition that biologic time and chronologic time are not the same. Obviously, therefore, chronologic age and biologic age are not necessarily identical, for the major variable in aging is time. They may coincide occasionally, but this is the exception rather than the rule. Each and every organism has its own physiologic age, which may be greater or less than its chronologic age. Furthermore, no organism is of uniform physiologic age throughout, for different structures and systems age at different rates at intervals in the life span. At certain periods involution of certain structures is accelerated, others may be temporarily retarded in their senescence. Physical infirmity may long precede mental senescence, there is many an old man with young ideas. In others, intellectual ossification may be far advanced before the organic skeleton shows the rarefaction of senility. Heredity, the mode and rate of life, accumulated injuries from the innumerable chemical, psychologic, and physical insults to which we are all subjected are elements which effect the biologic age. Aging, which continues throughout the span of life, progresses far more rapidly in the beginning than at the end, *the rate of change* (aging) is greatest in the fetus, less in the infant, still less in the child and after maturity becomes so slow as to be perceptible only by observations spaced widely apart. About 99 per cent of growth potential is used up during the intrauterine phase of development. The pathogenesis of senescence starts far earlier therefore, than we like to think.

The trajectory of human life follows a well defined pattern. Grossly divided into three periods, evolution, maturity, and involution, by Warthin,⁷ there are several obvious subdivisions of these phases. Intrauterine life is divided into embryonic and fetal stages. Postnatal evolution recognizes the periods of infancy, childhood, puberty, and adolescence. These are none too sharply demarcated, no exact measure is possible by chronologic age alone. The normal rate of development varies within fairly wide limits. From maturity onward, however, the subdivisions are even less distinct. The haziness of this part of the curve of change is due in part to the fact that the effects of aging become less and less obvious and are much more gradual in their evolution and also because of past lack of clinical interest and study of the *age factor in health and disease in adults*. The manifestations of aging are conspicuous and dramatic in infancy and youth. Aging change, and aging *per se*, proceed at ever diminishing rates, though the deceleration is not constant.

The various specialized cells of the complex mammalian organism do not all age at the same rate. Cells which divide frequently, such as those of the basal stratum of the epidermis, are very short lived, their individual span of life is from one mitosis to the next. Others, as for example those of the central

nervous system, live as individual cells as long as the organism survives. Between these two extremes are many variations, not only in the specific cell types or tissues, but in functional units, organs, or systems.

The ductus arteriosus goes through the involutional atrophy of old age while the organism is still very very young, bronchial clefts atrophy during fetal life. Thymic involution or senescence normally occurs in infancy. During the climacteric there occurs a relatively abrupt involutional atrophy of the organs of reproduction, even though other structures reveal no particular acceleration in their rate of senescence at this time. The placenta at term is perhaps the most striking example of senility coexisting with youth in the same organism. At the end of gestation the placenta, less than nine months old in chronologic time, is a senile structure, exhibiting all the histologic changes of old age. Yet it is intimately associated with a young uterus and younger fetus! This coincidence of evolution (growth) and involution (atrophy) occurs at all ages, though

TABLE I

Biologic landmarks	Average chronologic age at onset period*	Period
Conception		Embryonic
		Fetal
Birth	(9 months \pm 2 weeks)	Newborn
		Infantile
		Childhood
Puberty	(12 years \pm 2 years)	Adolescent
		Youth
Fertility	(19 years \pm 3 years)	Young adult
		Maturity
Climacterium	(45 years \pm 5 years)	Later maturity
		Senescent
Old Age	(70 years \pm 10 years)	Senile

* Note increasing variability in chronologic age at onset of various periods of biologic age.

the relative intensities of the two opposed trends vary with the composite age of the individual and with specific tissues of the organism.

Because of the conspicuousness of change in relation to age early in life the specialty of pediatrics developed. The growth of knowledge in this sphere of medical science has been phenomenal in the last fifty years. Pediatricians were the first to see and study the relation of age to health and disease. As a group they are the most conscious of all clinicians of the problems of gerontology. It was thus soundly logical, though superficially paradoxical, that Jacobs, the pioneer pediatrician, at ninety wrote the foreword for Nascher's pioneering volume on geriatrics⁸ (Nascher introduced the term "geriatrics" in 1909). The biologic and clinical problems at the two extremes of the life span, infancy and senility, are not dissimilar, in some respects they may be mirror images, except in rate and degree. The variability of biologic time over the spread of the curve of life is obvious. The nine month old infant has doubled its age at birth. Aging progresses far more rapidly in infancy and youth, the year from fifty-nine to sixty is but one-sixtieth of life. These smaller fractions of the whole, however, may become increasingly precious. We must not forget that psychologic time need not parallel physiologic time, emotion-

ally or intellectually The years slip through our fingers with increasing speed as the thread becomes frayed

One of the most important and fundamental contributions of pediatrics was the discovery and clarification of the fact that the child is not merely "the little man," but presents structural, functional, chemical, metabolic, nutritional, and psychologic *characteristics peculiar to his biologic age* This basic concept is equally applicable to geriatrics It is, in fact, the cornerstone of the growing structure of knowledge concerning aging and the aged The elderly are not just "old people", they are structurally, functionally, and mentally *different* men and women than they were in the days of their youth and early maturity The innumerable changes of senescence are continuous, though so slow and gradual that they are not measurable except by contrasting studies

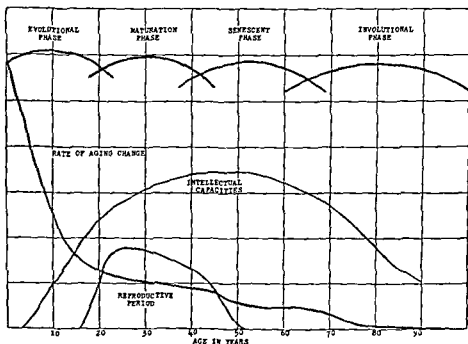


Fig 1 Approximate rate of aging in relation to life span of man

made at considerable intervals Wishful thinking encourages the delusion that one is still young whereas, in truth, the changes which lead ultimately to senile infirmity are already started We cannot stop aging To do so would be to terminate life But there is a hope that its consequences may be modified, particularly by control of the degenerative diseases which characteristically accelerate the changes

In studying first what aging does, we may divide the methods of approach into those researches concerned with structural change, functional change, and chemical change *Structural changes* include the gross changes in tissue which occur with aging, the histologic changes of specific tissues, the structural alterations in whole organs or systems, not only in man but in lower forms of life, such as the experimental laboratory animals, protozoa, plants, and even bacteria, for aging involves all forms of life ³ The structural changes

may be studied not only by the more routine methods of histology and microscopic pathology, but by histochemical methods which may reveal qualitative and quantitative changes in the lipoids, electrolytes, proteins, and water within the cells.⁹

Structural Changes. The fundamental anatomic alterations brought about by senescence *per se* are an atrophy of parenchymatous tissues and a corresponding increase in the amount of interstitial tissue (See Chapter 4.) The line of demarkation between the changes of so-called 'normal' aging, or the passage of time alone, and the pathology of chronic progressive disease states is so indefinite that no sharp distinction is possible. But as life without exposure to injury would not be life at all, but merely existence, it is truly academic to seek rigid definition as to where the normal ends and the abnormal begins. Furthermore, it must be emphasized that structural changes are not necessarily associated with functional depreciations of equivalent magnitudes or vice versa. The normal functional reserves are so immense that there are wide margins of safety despite the structural scarring of age.

Parenchymal cell atrophy has many distant consequences. Renal, hepatic, pancreatic, or thyroid atrophy and functional depreciations affect the whole organism. How much atrophic changes in one structure are dependent upon functional, chemical, and hormonal changes elsewhere is still unknown,¹² though evidence does exist that such interactions are significant (See Chapters 4 and 16).

Histologic and histochemical studies of individual tissue cells, whether parenchymal or interstitial, do not bring us much closer to the final mechanisms of senescence.^{13, 14} For though cells are the ultimate unit of life even in the most complex organisms, cells simply do not become old. With the single exception of certain cells of the central nervous system, the cells of mammalian tissues are constantly being replaced by young cells. Unicellular organisms do not reveal any of the structural changes which we relate to senescence, before they become old they divide into two young daughter cells. The tissue cells of man have but brief life spans. When the rate of replacement exceeds the rate of necrocytosis, growth occurs, when necrocytosis exceeds proliferation, atrophy follows. With aging there occurs a gradual shifting of the relative emphasis of these two processes. Though cellular proliferation continues throughout life, even unto extreme age, its rate relative to necrocytosis declines steadily.

Tissue culture studies, most notably those instituted by Carrel, have demonstrated that under proper nutritional conditions, cells are virtually immortal. Growth of cells from a chick embryo heart was maintained for some thirty four years with no abatement in the vigor of growth or change in the structural or biologic characteristics of the cells. This is several times the life span of even the healthiest chicken. However, and this appears to be the crux of the matter, such continued vitality is possible *only* if the culture medium is ideal and completely refreshed every twenty four to forty-eight hours. Immortality thus requires a continuously ideal environment. The quality of the environment is the determining factor, whether cells live in a tissue culture tube or in a living functioning organism. Thus we are brought face to face with the fundamental importance of the internal environment and of homeostasis, not only to normal physiology, but also to the phenomena of senescence.

Physiologic Balances. Functional changes of aging must be viewed from

both qualitative and quantitative angles. There may be change in either of these aspects of functional capacity and in the functional mechanisms. The internal environment of an organism is maintained at extraordinarily constant conditions. Thus we are able to determine a number of so called "physiologic constants," which are not really constants but are ranges within which variation is permissible without injury to the organism. For example, we may speak of such measurable phenomena as the pulse rate, the arterial tension, the body temperature, water content, and the concentration of chemical substances in the body fluids as constituting physiologic constants. These change to some degree with aging but we do not yet know just what proportion of these changes is in relation to normal physiologic senescence and what proportion may be due to the accumulative effects of previous disease. There is great need to know what the "normal" levels of these approximate constants are in relation to age. Normality is not a fixed point, but a series of variables. Before it is possible to make any evaluation of physiologic age and to say whether senescence is unduly rapid, normal, or delayed, we must know the *normal pattern of change* in these physiologic balances.

Though the constant may remain stationary, irrespective of age, this is not proof that there has been no *alteration in the mechanism* maintaining such equilibrium. Each equilibrium has its own mechanism for maintaining balance.⁹ These may vary with age in their speed of response and effectiveness even though the constant is not appreciably altered except under conditions of stress. For example, we know that the normal body temperature is essentially the same at all ages in normal man. However, the aged are very much more vulnerable to changes in external temperature and their mechanisms for maintaining a uniform internal thermal level become increasingly ineffective. The aged tolerate cold poorly, metabolic and vasomotor responses are much less effective than in youth. Similarly, the aged tolerate a hot environment badly, vasomotor reactions are less efficient and perspiration for heat loss is often inadequate because of atrophy of sweat glands. Again though the concentration of glucose in the blood of normal persons does not change notably with advancing age, there is considerable evidence to indicate that the ability of the older organism to withstand either an acutely increased absorption of sugar or a deprivation of glucose is decidedly decreased. (See Chapter 14.) Similar depreciation occurs to the mechanisms maintaining the hydrogen ion concentration of the body fluids. The hydrogen ion concentration of the blood is one of the so called physiologic constants which varies within a very narrow range, fluctuations beyond this range lead to immediate and serious disturbances. (See Chapter 37.) *It does not vary with age, but the ability of the homeostatic mechanisms to maintain equilibrium does become impaired with senescence.* This is revealed by the increasing ease with which acidosis, and particularly alkalosis, may be induced in the aged.¹⁰

Circulatory equilibrium, both in connection with the heart (see Chapter 24) and the peripheral vessels (see Chapter 29), is affected by age. It is fundamental that all the homeostatic mechanisms are slowed and less vigorous. Repair following injury is slower, changes due to disease are more frequently irreversible.

Responses to External Stimuli. Other functional attributes which change with aging are the physiologic responses to external stimuli. Study of such reaction must include the whole field of pharmacology, the response to phys-

ical agents, and the science of toxicology. There are immense areas in these sciences yet to be explored in order to reveal the relationship of physiologic age to the character and degree of response to such external stimuli (See Chapter 2.) Particularly important from the clinician's point of view will be those studies dealing with the consequences of mild but long-continued exposure to potentially noxious agents, such as may be part of our modern industrial civilization, i.e., the effects of smoke, dusts, industrial solvents, fumes of innumerable sorts, and many other as yet undetermined chemical insults to which mankind is being subjected by our highly artificial civilization. The margins of safety are reduced.

Chemical Approaches to Senescence Chemical, and particularly biochemical, approaches to the problems of senescence overlap to a great degree the field which is most commonly spoken of as physiology. The difference is only in the methods employed. Biochemical studies of changes of metabolism, cellular nutrition and respiration, and of growth in relation to age are needed. As chemistry is the study of the transformation of matter and as living depends upon the transformation of matter (metabolism), it is highly probable that the basic problem of the biologic transformations of aging will ultimately be solved by chemists. In future studies of the relationship of age to the chemistry of growth it is possible that the answer to the riddle of cancer may be found as a by product, for the problems of neoplastic growth are part of the greater problems of gerontology.¹¹

Theories of Aging Philosophic science does not wait for all the data before evolving theories. It is well that this is so, for theories provoke argument and differences of opinion and thus supply motivation for much energetic research. Competition enlivens thought as well as industry. One group works hard to prove a theory right, the other labors furiously to prove it wrong or, better yet, bring forth factual proof of a rival and opposing theory. Such has been the history of much of the dramatic advance in the biological sciences, and so will it be in gerontology.

The two major schools of thought are diametrically opposed. One concept postulates that involutional degeneration results from exhaustion and depletion of vital reserves by work and the wear and tear of existence. In opposition is the theory that the atrophies of senescence are due largely to disuse—that structural involution follows diminution or cessation of functional work. The known facts support first one and then the other theory, neither is proven. The balance of data for these two opposing concepts is so close at present that preference for one or the other becomes largely an emotional matter. Personal prejudice based on character, makes the choice. The lazy personality will instinctively favor the theory of exhaustion, rationalizing his indolence as "conservation of vital energy," whereas those filled with drive and enthusiasm will lean toward the second concept, citing the lives of those who achieved longevity by continued striving, only to decline shortly upon abrupt retirement. Let us not forget, however, that there are vast differences between use and abuse.

Our present concepts incline in the direction of primary changes in the interstitial tissues. The matrix surrounding parenchymal cells consists largely of fibers and fluids which are not themselves actually alive, but are nevertheless absolutely essential to the proper nutrition of parenchymal cells. It is upon these interstitial structures that parenchymal cells depend for their

nourishment, including oxygen, and for the removal of metabolic debris. Accelerated senescence, as manifested by arteriosclerosis, fibrosis, cirrhosis, and the like, is perhaps nothing more than an exaggeration of the aging of the colloidal gels which make up the fibrous elements of interstitial tissues. It is for this reason that serious consideration of Bogomolets' theories is warranted, though there is ample reason to be skeptical of all the claims made for his antitreticular cytotoxic serum.¹⁵ The relationship of the common chronic and progressive diseases of later maturity to cellular nutrition and therefore to the biology of senescence¹⁶ will be discussed later. (See p. 34.)

Abuse certainly accelerates aging changes. Abuse should include both excessive stresses and disuse as endogenous factors as well as the more obvious exogenous factors causing injury and premature depreciation. The clinical implications of these basic biologic concepts are discussed elsewhere in this chapter (see pp. 15 and 30) and throughout the whole book.

SOCIOLOGIC GERONTOLOGY

Sociologic gerontology concerns man as a social organism, existing in a social environment and being affected by it as well as affecting it.¹⁷ As cells are the units of which the structure of the human body is composed, so are individual men and women the basic units of the body politic. It is this aspect of gerontology which is becoming so acute that the need for intensive study is a matter of true urgency.¹⁸ The world is aging quickly, shifts in population structure have been accelerated in the last decade. Exhaustive discussion of statistical data is unnecessary here, a few figures will suffice.

Population Changes. Historical studies reveal that at the time of the Roman Empire life expectancy at birth was approximately 23 years.¹⁹ During the next nineteen centuries the average rose but very slowly. In 1850 the average life expectancy at birth was 40 years in the New England States. In 1900 this had risen to 49 years for the United States as a whole. Since the turn of the present century, the rise has been dramatic, in 1930 expectancy was about 60 years for white members of the American population, in 1940 it was 63 years. In 1945 the average length of life in the United States was 65.8 years, having increased over 16 years in less than half a century. Statistical studies²⁰ indicate that within the next decade or two it should be possible to extend the average life span to about 70 years.

Studies of the 1940 Census reveal that the median age of our population increased from 26.4 years in 1930 to 28.9 years in 1940. This is an increase of 2½ years within a single decade. It is estimated that continuation of the present trends will lead to a median age of approximately 40 years in the next half century. The numerical increase in the elderly is far greater than most of us realize. The total population of the United States increased 7.2 per cent in the ten years from 1930 to 1940, whereas the number of persons 65 years or older increased 35 per cent in the same interval. Certainly this relative rate of increase of those over 65 (almost five times the general increase) should constitute sufficient reason for affirming without apology that the problems of gerontology are of urgent importance. There are now approximately fourteen million people of 60 or more years of age in the United States of whom ten million have survived to age 65 or more.

Most of these gains are due to reduction in infant mortality. But there is also some increased expectancy in later years. In the United States the average

expectancy for women aged 40 in 1945 was 34.4 more years (expectancy to age 74.4), for men aged 40 in 1945, expectancy was 30.6 years (to age 70.6). For those aged 65 in 1945, the men will average 12.2 more years of life, the women 14.3.

The present situation is without precedent. Wars, famine, and pestilence have caused severe shifts in population structure but always in the direction of relative increase of youth. Such factors have all been destructive, the least fit failed to survive. Our problems today result from constructive forces. Medical science and the lessened hardships of modern civilization have enabled many less vigorous youths to survive into full maturity. The mounting millions of relatively unfit may jeopardize the survival of the most fit. Violation of a basic law of Nature, beautifully chivalrous though it be to assist the

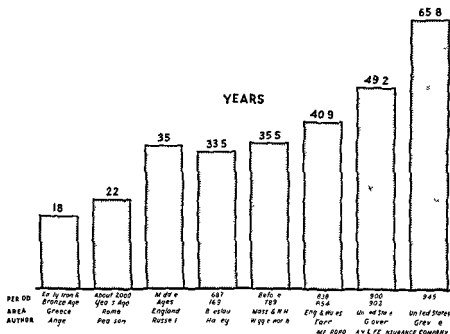


Fig. 2 Length of life

unfit to survive, may prove disastrously unwise. If society fosters research to save life it must accept the responsibility of the lives thus prolonged.

Social Medicine. The multitude of present and impending problems produced by these population shifts belongs to the realm of social medicine²¹ just as much as to the related fields of economics, politics, education, and religion. Social medicine must not be confused with "socialized medicine," which pertains to socialistic schemes of medical practice. Social medicine has been defined by Crew²² as medical science applied to *groups* of human beings. Social medicine deals with health *en masse*; we may contrast social medicine with clinical medicine by calling the former the wholesale approach, the latter the retail approach.

Social medicine is not a newly created discipline. The maiden, public health, has married sociology, and changed her name. Though altered by this

phenomenon of maturation and by learning from her consort, she carries with her all the hereditary and acquired characteristics of public health which we know so well.⁶ Let us hope this marriage is a fertile union.²¹ Social medicine takes the telescopic long range view of medicine which is so necessary to complete comprehension. It merely applies a different perspective upon problems of health. Social medicine and clinical medicine have identical objectives. They seek to approach their common goal by different routes and techniques. Both are concerned with health, the one individually, the other collectively or environmentally.²¹ As man cannot be isolated from his environment, it is inevitable that there are innumerable points of contact between the two disciplines.

Political and Economic Implications. The political implications of the recent dramatic shifts in the age structure of the population are alarming. The clamor of the aged for economic security may be heard throughout the breadth of the land. It will become louder. Fantastic and obviously unsound schemes are advocated with fanatic vigor and dangerous political skill. When politicians awaken to the implications of the rapid increase in elderly voters even worse situations may arise. Socialistic schemes, pampering paternalism by the state, and financial assistance for the elderly at the expense of younger age groups will not solve these problems. The personnel policies of industrial management, the economics of life insurance and health and accident insurance underwriting and of banking, the character of future advertising programs and the design of consumer goods, and retail merchandising methods all face the necessity of decided revision as the nature of the nation changes. These are but some of the problems facing our economic order in relation to aging. Others, equally urgent, involve education. The leaders of religious teaching need to take cognizance of the increasing average age of their flocks and broaden their narrow interpretations to fit the more critical and more mature minds of the future. There is danger in neglecting to do this, for aging brings about fixation and intensification of character. The understanding and tolerant become more tolerant and gentle with age, the intolerant and sanctimonious become bigoted reformers with all the cruelty and injustice of religious fanatics. The mounting economic and social burdens of chronic and disabling mental and physical illness are appalling. It is obviously impossible to consider all the vexing problems here, but brief discussion is warranted of those social questions most intimately associated with medical practice.

Physicians can and must be leaders in working out tentative solutions for these complex and pressing socio-economic questions. Though as individual doctors we deal only with the individual units, men and women, which go to make up the body politic, the medical profession *en masse* is the one and only group in direct contact with people whose members have the requisite training in the disciplines of science to be effectively conscious of the effort necessary to remain objective. Advance toward solution of these problems will need all that both the science and the art of medicine can give. Statesmen who deal

of life. It is also the physician who must clarify and understand the changes in capacities, the limitations and the potentialities of normal senescents. The art of medicine is needed to discover these changes and to guide the elderly wisely according to their capacities so that their useful productivity is no longer wasted. Such knowledge is prerequisite to intelligent attack upon the socio-economic menaces of our aging population. It is the responsibility of geriatrics to acquire and to apply the necessary knowledge.

The Potentialities of Maturity. Depressing and alarming as these remarks appear superficially, the future is not all dark. There exists an immense and largely untapped reservoir of treasure in the elderly. Longevity with health and postponement of disability until true senility brings about infirmity *can* be made the basis for splendid advance. But it will require much effort. The science of geriatrics must learn to prevent or control progressive disease and the art of geriatrics must apply the knowledge thus learned. Wisdom is conditioned by age, for its development depends upon experience, in which time is a major factor. Let us not delude ourselves, however, into believing that aging creates wisdom. It merely permits it to develop in those intelligent enough to learn by experience and prepared to observe and profit by such learning. The young fool will become the old fool should he live that long. The bright lad should become a wise seer if his health is guarded and his mind is trained. It is not coincidence that the engineers of crack trains, the captains of the finest ships, and the leaders of industry, science, education, law, and of the State itself are old men. It is by reason of their judgment, specialized knowledge and laboriously acquired experience that the gravest responsibilities rest upon aging shoulders. Responsibilities of decision in organizing and mobilizing national resources into effective military might to protect our culture, more priceless than life, weigh heaviest on those well along in years. The central nervous system of a body politic is composed of older men and women, just as the cells of the brain are among the oldest in the body of the individual organism. Whether we wish it or not, these thinking units control our destiny. Conservation of the health and vigor of these precious and most difficultly replaceable elderly minds should be an essential part of our efforts for defending our culture.

There is great hope in the thought that as a culture (which is the composite judgment and sense of values of a race, nation, or group) ages it becomes wiser, finer, and more tolerant. We may hope that this era of increased longevity may lead to cultural maturation. Science is quickly exploring and exploiting the physical world about us, but it remains the privilege and the responsibility of *mature* men and women to reveal and develop the full potentialities of mankind.

CHRONIC DISEASE

Senescence is an important factor in the causation of many chronic diseases. These increase sharply in incidence with aging. They are not limited to the senile, the beginnings of chronic and progressive heart disease, arteriosclerosis, arthritis, diabetes mellitus, and the like occur with the beginnings of the major involutions of senescence. Characteristic of these "degenerative disorders" is their slow but inevitable progression, insidious onset, and long periods of disability. The toll of disability in the fifth and sixth decades is a graver economic problem than deaths from acute infections.²⁴ Brutal truth

forces one to confess that a man in his forties or fifties who will be disabled for many years by arthritis or coronary or cerebral arteriosclerosis is a greater social loss and heavier burden upon his family and society than a man quickly dead. Mortality tables alone are most misleading indices of the social significance of disease. The human wastage from disability due to chronic disorders is incalculable.

Data from the National Health Survey of 1935-36²⁵ show the importance of age in chronic invalidism (see Fig. 3). Over 1 per cent of more than three million persons studied were chronic invalids. The charts (Figs. 4, 5, and 6)

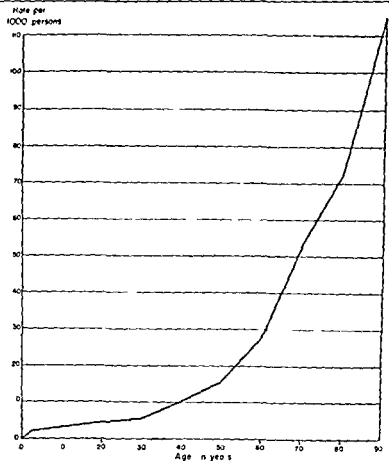


Fig. 3 Number of invalids per thousand population according to age (U S P H S)

emphasize the lack of parallelism between mortality, prevalence, and disability. Among the chronic disorders of later years, cardiovascular disease is preeminent as a cause of death,⁶ although "rheumatism" (the arthropathies) exceed this group in incidence. Allergic disorders, such as asthma and hay fever, are exceedingly prevalent, cause much intermittent and partial dis-

related and overlapping etiology (see p. 17)

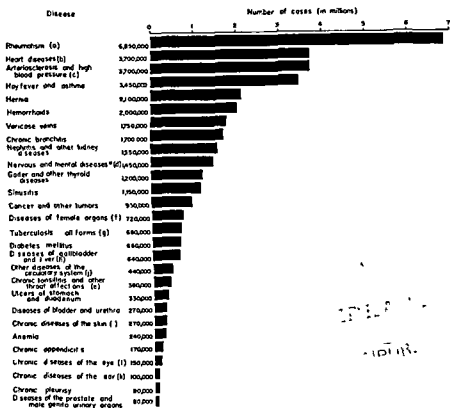


Fig. 4 Estimated prevalence of certain chronic diseases in the United States during 1937 (U S P H S)

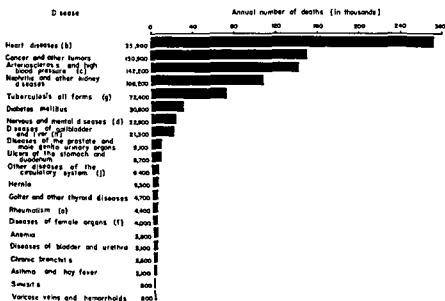


Fig. 5 Estimated annual number of deaths due to specific chronic diseases in the United States, 1937 (U S P H S)

The order of importance among chronic disorders in causing prolonged and/or progressive disability is (1) *mental and nervous diseases* (269,300), (2) *cardiovascular-renal diseases* (236,100), (3) the *arthropathies* (147,600, and (4) *tuberculosis* (77,900). Cancer accounts for but 28,100 disabled invalids, or about one-tenth as many as rendered economically useless by mental and nervous disorders or by cardiovascular-renal disease.

These comparisons, which need not be precisely accurate to be impressive, emphasize the asymmetry of present research efforts. Study of the problems of cancer and tuberculosis, and even syphilis, is liberally supported by both private and public funds. This asymmetry must be ascribed to emotional

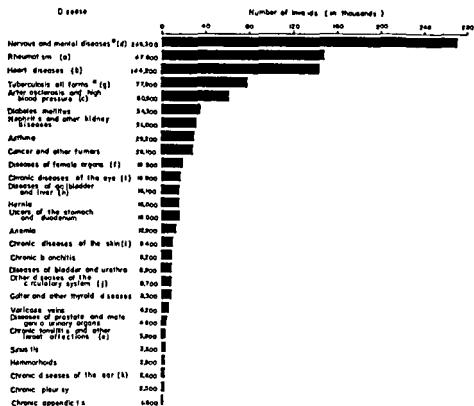


Fig. 6 Estimated number of invalids disabled by certain chronic diseases in the United States, 1937 (U S P H S)

factors, certainly logic is not involved. There is more drama and fear associated with cancer than with vascular disease.

Social medicine can do more than it is doing in obtaining and disseminating information regarding the urgency of research in certain areas. Such information is needed particularly by those determining the distribution of research funds and facilities, both public and private. A careful survey by

four dollars were spent for research into these disorders, for each death due to cancer, two dollars were invested in cancer studies, for each death due to

poliomyelitis, five hundred dollars were assigned to research, and for each death due to circulatory disorders only seventeen cents went for research in the field of the disease.

Arteriosclerosis, hypertensive disease and cardiac disorders, are predominant factors

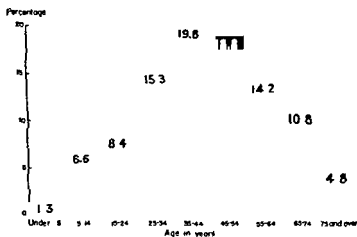


Fig. 7 Percentage distribution of persons with chronic disease or permanent impairment according to age groups (U S P H S)

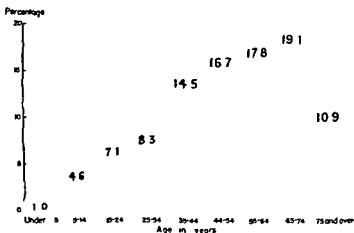


Fig. 8 Distribution of invalidism according to age groups (U S P H S)

in the pathogenesis of many of the mental and nervous disorders (see p. 271), and of the arthropathies (see p. 675) the relative urgency of the problems of

ment and invalidism are at their peak from 50 to 74. Obviously a huge fraction of the disability in this senescent age group arises from the progressive, but as yet not disabling disorders so common twenty years earlier. These data dra

matically emphasize the concept that the sources of geriatric disability arise in the thirties and forties. Therefore it is in the period of later maturity that preventive geriatrics may hope to be most effective.

The recent increase in admissions to mental hospitals because of disorders of senescence and, particularly the circulatory defects of later years, is truly startling. In the quarter century from 1912 to 1936 the admission rate increased from 7.7 to 49 first admissions to state mental hospitals per 100,000 of population over 40 years of age.²⁷ This rise in admission rate of more than 500 per cent cannot be attributed solely to increased frequency. Social and economic factors, greater crowding in urban living, changing attitudes toward hospitalization for the mentally sick, and a diminishing sense of individual family responsibility fostered by governmental pampering all contribute to this dramatic increase.²⁸ Nevertheless, the terrific economic burden of the mental invalids is here, it will further increase until such time as medical science may point the way to prevent or retard the vascular disorders so intimately associated with senescence.²⁹ The medical aspects of mental disease in the aging and aged are more fully and more competently discussed elsewhere (see Chapters 2 and 17).

INDUSTRIAL HEALTH

Industrial medicine began as an outgrowth from emergency traumatic surgery and then grew to include hygiene and the toxicology of industrial gases, fumes and dusts. It has only recently become aware of the potentialities of more personal preventive medicine in contrast to the wholesale protection of workers from undesirable environmental conditions. This awareness of the importance of the individual has been accelerated by studies into the sickness absenteeism of nonindustrial origin.³⁰⁻³¹ Sickness absenteeism of nonoccupational origin is responsible for many times as much lost time as disability due to industrial injuries of all sorts. Industry is in an exceptionally advantageous position to foster and maintain the health of its workers, a position parallel to the development of preventive medicine in general.

Because the industrial worker in the plant is under medical supervision approximately one third of his time, the industrial physician has splendid opportunities for education, if he will but avail himself thereof.

Industry is intimately concerned with the problems of aging.^{32, 33} Personnel directors for manufacturers are becoming conscious of the fact that the average age of their employees is increasing at a rapid rate—almost a year per year in one immense organization. The average age of workers will not diminish. Industrial medicine has two major functions in connection with the aging of employees. One is *diagnostic*, the other *therapeutic*. Employers must have certain basic information as to the physical condition of workers in relation to age before they may be wisely employed and placed. After such older workers are employed, management needs to know their physical conditions

retirement necessary. These decisions can be made only on the basis of objective medical data evaluating the health of employees.

The second function of industrial medicine is the *therapeutic* one. It must not be forgotten that operating departments in industry employ inspectors to find flaws and defects in equipment and also have service or maintenance workers to repair these defects and maintain equipment at the maximum of efficiency. Men are the most important machines or productive units in any organization.³⁴ Therefore, the periodic health examination should not only detect defects but should help to correct them. Diagnosis exists primarily for the purpose of treatment. Periodic health inventories with the data recorded on a card and quickly filed away are sadly useless.

The economic aspects of *continued employment of older workers* need little comment. Obviously, if men are to be laid off in their early fifties or even in their early sixties, the economic burden for the support of these individuals can but become disastrously oppressive upon the younger segments of the population. If these increasing millions of older individuals can be employed and support themselves with work suited to their capacities, the productivity of the nation will be augmented. The one solution is constructive, the other destructive.

With aging comes slowing of speed. It is often impractical to continue older men side by side with younger workers at the same rapid pace of modern industrial production. But aging brings about compensations, with wise planning these compensations could be utilized. It is not necessary that the aging mechanic or artisan, whose speed is reduced but whose skill and judgment are enhanced, be discarded or transferred to sorting nuts and bolts in the junk room, or to watching a gate. Such a transfer is equivalent to a judicial sentence: "You are through and useless, you may no longer feel pride in your work or have the priceless satisfaction of feeling really useful." All of us know instances in which premature retirement was but the precursor of the death certificate. The greater the skill, the finer the character, and the more ambitious the worker the more disastrous is such a blow.³⁵ Under such irreparable condemnations even the best men quit. When the will to live is gone the science of medicine is pathetically futile.⁵

Personnel organizations frequently spend much time and money in at-

definitely. The job remains the same, but the man does not. He changes with age. The fit between man and job becomes increasingly incongruous as maturation and early senescence alter his physical and mental capacities, his *perspective, his speed, interests, and ambitions*. From this increasing lack of fit between man and job arise many of the occupational neuroses that hasten the senescence of personnel management.

The potentialities of the senescent for service have barely been touched. Accomplishments of such men as Oliver Wendell Holmes, W. H. Welsh, Goethe, Edison, Titian, and many others in the evening of their lives are mere indicators of the vast storehouse of neglected treasure in those so often viciously dubbed "old men." Only in the relatively independent professions, such as medicine and the law, is the value of matured judgment from long experience given proper recognition.

EDUCATION

Preparation for senescence through education has not kept pace with the changing social order. College and secondary school curricula are still geared to the day when life expectancy was more than fifteen years less than it is today, when it sufficed that education attempt only preparation of the boy or girl for the competition of early maturity. Continuation of such attitudes places education about fifty years behind the times. There is not the slightest evidence that educators are aware of the need for preparation for senescence. The false and complacently smug assumption that the art of aging is learned spontaneously has grievously retarded development of the potentialities of the elderly. The child prepares to become an adult; the adult must likewise prepare for senescence if old age is not to be crabbed and bitter. Happy, full, significantly useful lives are not fortuitous. They are predetermined by preparation. It is high time that schools revise their objectives and recognize their obligations in the later years of life.

Greatness of mind in later years comes from *continued study*. This is rarely encouraged among adults, with the notable exception of professional groups. Doctors who become great continue to study, exercise conscious effort to learn. On the whole, however, adult education is in a deplorable state in this country today. Facilities are grossly inadequate. Personnel is uninspired and untrained to meet the special problems of pedagogy appropriate for adult minds. There has been altogether too much oversimplification.

Many years of repetition of an *incorrect dictum* eventually leads to its adoption as truth. The woefully erroneous concept that the *older mind* cannot learn has done immeasurable harm. Constant reiteration of such platitudes as "You can't teach an old dog new tricks" has so fixed this false statement in the minds of most aging people that they are *afraid to try to learn*. That the concept is false has been proved repeatedly by the studies of Miles, Thorndike and others (see p. 98). These investigations demonstrated that the *ability* to learn depreciates very slowly indeed if the *will to learn* is retained. Miles has pointed out¹² that the *increments of knowledge* accumulated through the years and the continued practice of mental exercise favor preservation of mental abilities. Critical judgment is enhanced by aging, superficiality and the gloss of pretty phraseology used to conceal ignorance are quickly discerned by the adult mind. In order to teach the old dog new tricks, it is necessary to know more than the dog.¹

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making continued, part time education available throughout life. If all but the dullest youths who pass on from formal schooling have not acquired the habit and hunger for continued learning, then education has failed in its obligations.

It is unfair and illogical, however, to place all educational responsibility upon professional educators. Example is still the first principle of successful pedagogy. Parents mold their children. These children become parents in turn, and, later, grandparents. Familial environment is a potent educational

ism produces perverted personalities and parasites, an environment of vigorous curiosity and parental desire for usefulness creates thinkers and doers. Every privilege carries with it responsibility. This is perhaps the greatest single lesson that parents can teach their children. *The privilege of longevity entails the responsibility of effort to maintain health and retain usefulness.*

Society today denies both youth and the aged the privilege of labor. Child labor laws, originally introduced to prevent undue exploitation of children have become political weapons of weak-minded but vociferous sentimentalists. Employers *must* require that so many birthdays have passed before they may hire youth. Enforcement of these laws requires a host of nonproductive

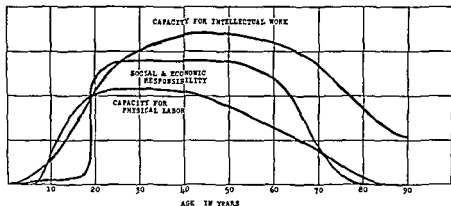


Fig 9 Relative curves of approximate capacities for physical and intellectual work in relation to age contrasted with usual extent of responsibility as permitted by present day society. The abrupt rise and nearly as sudden fall of the responsibility curve is decidedly not biologic.

government employees. But no one has attempted to assay the immense social damage done by depriving quickly maturing youth of the right to work. Parents assume that work is injurious. As a result, the youth of the nation is

crashes in the early twenties, and was such an important factor in the psychiatric problems of the armed forces in and since the recent war.

CULTURAL POTENTIALITIES

Thus far the hazards, the social liabilities, economic costs of chronic disease and premature unemployment have been emphasized to the unfortunate exclusion of the potential advantages of an older population. The potentialities of maturity have hardly been recognized, let alone explored or developed. We have hinted at the fact that the full potentiality of men and women can be attained only by those reaching ripe maturity. But what does this mean?

Aging, as has been pointed out, is asymmetric. As certain somatic and psychic attributes decline with aging, others become stronger. Memory may decline (see p. 98), but judgment in the appraisal of the significance of facts improves with age. Visual acuity obviously diminishes (see p. 296) but the ability to comprehend that which is seen improves with experience. Experience is dependent on time and therefore inevitably grows with age. The intelligence, as measured by the Bellevue scale, declines very slowly indeed in later years, in some respects qualitative changes compensate for the quantitative depreciation (See p. 97.) Many factors, such as motivation, interest, practice, or disease, alter and distort the quantitative studies on those in later maturity.

It is characteristic of the older mind that it becomes more and more interested in relating facts and concepts to one another and less interested in the mere accumulation of new factual data. The mature mind becomes analytical and critical and thus increasingly comprehending. No fact is isolated, it is related to all other facts though the relationships are often obscure. The mature mind is perhaps more concerned with the cement relating facts to each other than with the isolated facts. The relation of the past to the future grows in importance, the child is interested only in *now*, not tomorrow or yesterday. Integration acquires precedence over acquisition of new facts. Thus judgment becomes seasoned and strengthened.

However, it must not be assumed that aging alone suffices for the development of judgment and wisdom. There is no justification for reverence for the words of the elderly just because they are uttered by the elderly.

Emotional maturation does not necessarily parallel intellectual development. Frequently it lags far behind. Emotional maturity is truly a rarity. But, rare as it is, it is limited to those who have reached physical maturity. Thus the preservation of those chronologically and biologically mature is necessary for the development of an emotionally mature population. And, as Chisholm has pointed out,³⁶ enhanced emotional maturity is the prime requisite for a world at peace.

SUMMARY

It has not been possible to present more than a resume of the problems of aging and the aged here. The immensity of the field of gerontology would require many volumes for comprehensive discussion. It is hoped, however, that the foregoing may serve to illustrate some of the many unanswered questions, provoke curiosity for further study of some of them, and reveal the intimate correlation of the three major categories of thought involved. Long range orientation must precede and accompany specialization if narrowed vision and asymmetry of emphasis are to be avoided.

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The present volume is concerned with the second division of thought—the problems of aging men and women. Geriatric medicine must not be construed as being limited to the clinical problems of the truly senile, the disorders and functional and structural changes of senescence start long before senility. Understanding of the processes involved is prerequisite to full comprehension of the consequences of aging. From the viewpoint of preventive geriatrics the

CHAPTER 2

PRINCIPLES OF GERIATRIC MEDICINE

EDWARD J. STIEGLITZ

GERIATRIC medicine is that branch of medical practice which deals with the *clinical application* of scientific knowledge to the medical needs of the aging and the aged. It is the third perspective of gerontology—the “naked eye” approach. Within the present chapter we intend to discuss certain basic principles, to be further elaborated elsewhere in the text. As the unit of clinical practice is the individual, we must remember that any efforts at subdivision are arbitrary and artificial. Modern medical practice has become so complex and demanding of such highly specialized skills that clinicians have tended to become more and more specialized by reason of technic (surgery vs. internal medicine) or anatomic areas (ophthalmology, neurology, orthopedics, urology, dermatology, and the like). Geriatric medicine crosses the lines of all these various structurally delineated specialties.

The focus of attention in geriatric medicine must ever be upon the individual patient, his peculiarities and significant characteristics, be they structural, functional, or psychologic. We are more concerned with the individual and his welfare than with his disease.

SCOPE

It must be emphasized that the problems of geriatrics are not limited to the actually senile any more than the problems of pediatrics are limited to those of the newborn infant. Any demarcation of these various fields based solely upon chronologic age is sure to be arbitrary and artificial. There is no complete agreement as to just when pediatrics ends, nor can there be unanimity as to just when geriatrics begins. If geriatrics were limited to the study and

begin many years before. They actually begin in infancy but are rarely significant before the peak of maturity. Therefore, it is pragmatic to consider that the majority of the problems peculiar to geriatrics *start* at about forty years of age, the approximate meridian of life. It must not be assumed that all those beyond forty are decrepit and infirm by reason of senility! But it is a logical working hypothesis that the involutional changes leading to later infirmity insidiously accumulate from approximately this age onward. If during the age period from forty to sixty health may be maintained near the optimum and the so-called degenerative diseases (particularly the cardiovascular disorders) either be prevented or their progression much retarded, then the likelihood of long disability, chronic illness, and uselessness after sixty will be immensely reduced. Obviously far more may be accomplished for the aging than for the truly senile aged. If geriatrics is to be really effective, an attitude

of prophylaxis throughout this critical period is the most important stone in its foundation¹

Geriatric medicine must consider both normal aging men and women and the abnormalities or diseases characteristic of the senescent period, just as pediatrics is concerned with well and sick infants and children. In both of these fields of practice, special problems arise out of the basic fact that *aging brings change*. It is at the beginning and at the end of the life span that the manifestations of the changes introduced by aging are most conspicuous. Nevertheless the processes are continuous and the rate of change does not necessarily parallel the obvious clinical manifestations. One of the most fundamental concepts of pediatrics is that children present structural, functional, metabolic, immunologic and personality characteristics peculiar to their age. This concept must likewise be the foundation upon which geriatrics may build a scientific approach to its problems. The elderly are not just "old people," but are physically and mentally different men and women from what they were in the days of their young maturity. The changes of the senescent period, just as those arising during the period of development, do not proceed at the same rate in different individuals, nor do they progress uniformly throughout the organism.

Aging, as previously mentioned, involves two antagonistic types of change—evolution or growth and involution or atrophy. These occur concurrently throughout the life span, though their relative intensity and rate vary. Obviously, during infancy and youth growth predominates and, equally obviously, during senility involution predominates. Despite the conspicuousness of growth phenomena in youth, it must not be assumed that atrophies are absent. Illustrative examples include the involution of the branchial clefts

there are certain growth phenomena associated with senescence which are of the greatest medical importance. The innumerable types of neoplasms, whose etiology is in some occult fashion associated with aging and which tend to arise during senescence, reveal the potent residuum of the growth force in elderly organisms. The changing pattern of relationships of these two opposing forces, growth and atrophy, in all the many complex functions and structures of the body is responsible for some of the peculiarities of medical practice with the elderly.

Involutional changes do not become dominant until at or after the peak of maturity, at approximately forty years of age. From this age onward occur the insidious involution phenomena whose accumulation later creates the consequences of senility. It is, furthermore, approximately at this age that the so-called degenerative disorders become conspicuously increased in frequency.

Thus we may say that geriatrics should begin at approximately forty. The span of life after the peak of maturity may be divided into three phases: the period of later maturity (approximately forty to sixty), the period of senescence (approximately sixty to seventy-five), the period of senility (after seventy-five). It is immediately admitted that the demarcation of these periods by chronologic age is open to controversy and that there are many exceptions. Some persons are senile long before seventy-five. There are, however, others who should be classed as in their late maturity even after sixty. Grouping of

the problems of geriatrics into these three periods is purely a utilitarian measure to define arbitrary lines of demarcation. In each one of these periods there are certain characteristics of the organism which profoundly alter clinical practice. The triad of phases may be paralleled by the four periods recognized by pediatrics: newborn, infantile, childhood, and adolescence.

OBJECTIVES

Having thus attempted to define the scope of geriatric medicine, it might be wise to consider its objectives. These have often been confused. There are many who resent the implication that geriatrics should attempt to prolong life. Many among the senile, and even more among the young, feel that most of the older members of our population are living too long as it is, that the vastly increasing numbers of the old are becoming naught but a burden upon the community. These soured egoists assume that the primary objective of geriatrics is to prolong life. Their assumption is erroneous. Equally in error are those who would assume that the sole objective of geriatrics is to relieve suffering in the elderly. Such would be a most negative and defeatist approach. Geriatrics can and will aim further than this.

There can be no desire to arrest aging or to prolong greatly the life span. The life span of any species has definite biologically determined limits. To arrest aging would be to terminate life. Rejuvenation is a myth begotten of wishful thinking. The primary objective of geriatrics recognizes these limitations and seeks to modify the abnormalities characteristic of senescence, and to control the progressive disorders of later years, thus *adding to the health and usefulness* of the elderly. There has been no better definition of the major objective of geriatrics than that of Piersol and Bortz.² "The society which fosters research to save human life cannot escape responsibility for the life thus extended. It is for science not only to add years to life, but more important, to add life to the years."

Therapeutic Approach. Therapy may be classified on the basis of objectives as well as technic. Of the five therapeutic approaches which are available to geriatric medicine, prophylactic, control, and constructive therapy are the most significant. An attitude of prophylaxis, of anticipation of future difficulties and progressive change must permeate geriatric therapeutics if it is to accomplish all that it is capable of accomplishing.

Prophylactic Therapy. Prophylaxis has come to mean the prevention of

cision and completeness of knowledge concerning the etiology of any given

possible to immunize individuals against hypertensive disease or to protect them from diabetes mellitus by quarantine. In order to prevent, it is first necessary to know most of the causative factors.

Curative Therapy. Possibilities for cure in geriatrics are likewise affected by our ignorance of etiology. Cure of disease is predicated upon eradication of the causes thereof. Furthermore, cure is obstructed in the geriatric patient by the diminution of the capacity of the senescent organism to repair itself.

after injury. Any advance in knowledge of etiology can but be reflected by great advance in effectiveness of therapy.

Palliative Therapy. Palliation seeks to conceal distressing symptoms in an otherwise hopeless situation. In the truly senile palliative therapy is often all that we can hope to apply. In the intermediate age groups, late maturity and senescence, therapy for control is particularly pertinent.

Control Therapy. It is curious that there is no adjective for the noun control. "Controllative" is not in the dictionary, though it should be. "Controllative" therapy seeks to reestablish homeostasis when disturbed by disease and also to retard progression. These two goals are often attained by the same mode of attack, for anything which corrects imbalance must postpone the consequences of such disequilibrium. For example, it is well established that uncontrolled diabetes mellitus progresses far more rapidly than controlled diabetes mellitus (see p. 218). Similarly, the pathologic changes in the arterioles in hypertensive arterial disease progress more quickly in the uncontrolled hypertensive than when appropriate management diminishes the continuous vascular hypertonia (see p. 468).

Constructive Therapy. This is a relatively new approach in adult medicine.³ It differs from prophylaxis inasmuch as the latter is directed toward preventing specific diseases, whereas the former attempts to raise the level of health. Constructive medicine includes preventive measures but goes beyond these to foster optimum physiologic efficiency.⁴ The applications of constructive medicine will be discussed in Chapter 6.

A basic principle of geriatric medicine is the dictum that more can be accomplished for the aging than for the aged. When senility truly equals infirmity there are no miracles which turn back the hands of the clock or reverse the direction of time. Senility is the end point, the consequence, of senescence. Control and careful constructive guidance through the period of senescence, however, may retard and diminish the depreciations of aging. Thus a continued attitude of prophylaxis is essential to the full development of the potentialities of geriatrics.

Factors Affecting Geriatric Medicine. Two sets of factors influence all aspects of geriatric medicine: (1) characteristics of aging peculiar to the individuals involved and (2) characteristics of the disorders most commonly encountered in the senescent period of life. On the one hand we must consider the patient who has a disease, on the other the disease which is making the patient ill. Certain characteristics of both patients and disorders affect geriatric diagnosis, prognosis, and therapy more than others. Many of these attributes will be discussed with considerable thoroughness in subsequent chapters. Here we wish to emphasize and correlate the more significant characteristics of widespread importance.

SIGNIFICANT CHARACTERISTICS OF SENESCENT PATIENTS

Individual Variation. With advancing age there occurs an increasing divergence between individuals. We are today what we are largely because of the experiences of our yesterdays. The older we become the more yesterdays have affected us. No two people experience the same injuries, infections, nutritional insults, intoxications, fatigues, or emotional traumata. In no two instances are these inevitable vicissitudes of existence identical in character,

in severity, or in sequence. Aging *per se* is asymmetrical and variable. As pointed out previously, biologic age is not uniform throughout a given individual and thus the physiologic efficiency of various structures is affected asymmetrically by senescence alone, and, of course, even more so by the scarring of the inevitable and innumerable minor and major insults to which everyone is subject if he lives long enough.

Individualization is an absolute necessity for effective application of clinical geriatric medicine. Routines should be avoided as not only undesirable but dangerous in inviting errors in diagnosis, therapy, and prognosis. Each and every older patient is a problem unto himself or herself, differing from all other patients to a degree that warrants constant consideration. A careful, detailed, and patiently explored past history is the primary step in evaluating the individual. We should be concerned not only with what diseases have occurred in the past, their severity, sequence, and evidences of sequelae, but also with habits, marital experiences, occupation, education, familial background (from both the viewpoint of biologic heredity and that of childhood environment), diet, and the like. All these factors operate to affect the individual as he exists today.

Variations involve not only structural differences and somatic functions but also the psychologic pattern or personality. Such variations are significant in both diagnosis and therapy. Unless the emotional characteristics are appreciated and advice so presented that it can and will be followed, little benefit can result, no matter how sound the advice may be. Superficial reassurance will not suffice for the apprehensive neurotic, deeply afraid of possible cancer, dogmatic restrictions without statement of valid reasons will be ignored by the aggressive individualistic hypertensive. In later years successful therapy depends more and more upon the efforts of the patient and less upon medication or imposed procedures. Therefore, intimate and continued cooperation between patient and physician is vitally necessary for good therapeutic results. Obtaining and retaining such cooperation depends largely upon individualization in therapy, "production-line" or routine methods simply will not work.

Accumulated Injuries. As just mentioned, living involves a long and varied series of insults and injuries. Many of these are unavoidable except by existence in an isolated, artificial environment. Such would not be living, merely existing. Some injurious experiences are necessary for the development of protective capacities, certainly the reaction to typhoid or smallpox vaccine is less detrimental than an attack of either typhoid fever or smallpox. One cannot develop courage without having experienced fear, nor muscular strength without hard work. Yet every toxic, psychic, or traumatic experience leaves some residue of scarring behind. The detriment may or may not be apparent or even detectable. Revelation of the consequences may be long delayed. For example, we do not actually see the cloudy swelling of parenchymatous tissues associated with fever, though we know it occurs and that subsidence of the acute febrile intoxication does not imply immediate or perfect repair of this damage. Every such injury leaves some minor residue. Individually they may be negligible, but their accumulative significance is considerable. Even pathologists are unable to distinguish which structural changes in senility are due purely to aging and which result from accumulative injury. (See Chapter 4.)

Diagnosis is greatly complicated by the existence of previous injury. It is frequently extremely difficult to differentiate the origin of observed clinical

phenomena (symptoms and signs) in an older patient especially if seen for the first time in an acute illness. Evidences of disordered function may be due to preexistent depreciations or to the acute malady superimposed upon a previously asymptomatic impairment. Yet it is most important to know how much of the disorder is new (acute) and how much preexistent (chronic). An acute illness may so exacerbate a previous condition that the latter takes on the characteristics of another acute affair. For example, the mild diabetic may be symptom free and unaware of diabetes mellitus until an acute infection so lowers the glucose tolerance that the diabetes becomes obvious. It is but natural for many to assume that the acute infection caused the diabetes, though this is clearly incorrect. Similarly an acute bronchitis may be superimposed upon preexistent but nearly asymptomatic tuberculosis with great distortion of the physical findings, x ray changes and clinical course. It is axiomatic that it is much more difficult to discover or evaluate a chronic disorder in the coincidental presence of an acute disease than prior to the acute affair. Tuberculosis is far more common among those over fifty than most physicians realize,⁵ to be discovered it must be searched for. In a recent x-ray survey of 114 500 persons in New York State, the incidence of probably active, probably inactive, and suspected tuberculosis in those past forty five was decidedly higher than in those younger than this age. The percentage for all ages was 1.2 per cent, for 45-54 it was 1.9 per cent, for 55-64 2.8 per cent, for 65 and over 3.1 per cent.⁶

Prognosis is likewise greatly affected by the existence of preexisting lesions of any sort. In many respects the state of health, or the degree of depletion engendered by previous injuries, is as significant as the active therapy administered in determining the outcome of an acute illness in those past the peak of maturity. The importance of having a *known base-line*, of knowing the functional and structural status of aging patients prior to acute illness, should be obvious. It is only in this manner that we can with certainty distinguish new and preexisting deviations from the normal. Here as elsewhere, to be forewarned is to be forearmed.

It is in connection with prior accumulated injuries that geriatric medicine stands in sharpest contrast to pediatrics. The pediatrician has the right to assume that prior to an acute illness the child was fundamentally well and that therefore all the symptoms and signs observed are due to the acute disorder. In dealing with older patients we must assume precisely the opposite—that prior to the acute illness the patient was not in full health and that therefore many of the clinical phenomena observed may be consequent to preexisting conditions.

Impaired Homeostasis It is well known that the internal environment of the organism must be maintained at nearly constant conditions. This internal milieu is not grossly altered by aging. The ranges of almost all the known "physiologic constants," such as temperature, pulse rate, respiration, concentrations of same at eighty
to maintain at

adaptation lowers the margin of safety when either external or internal conditions tend to disturb the delicate balances which mean health

Early lowering of homeostatic efficiency is revealed by the changing responsiveness to stress. Acute illness may create such stresses, a break in tolerance or decompensation may thus lead to the creation of confusing complicating phenomena which are secondary rather than primary consequences of the disease. Stress may be induced deliberately, as in some of the stress functional tests, in order to reveal early depreciations. The importance of these clinical stress tests in evaluating physiologic age or the degree of health will be discussed more fully in Chapter 6. Because of the immense functional reserves of all biologic functions, conditions of increased physiologic load must be created to reveal depreciations before they become so extreme that functional failure exists at nearly resting levels.

Three clinically significant characteristics of older individuals are derived from the slowing and weakening of the homeostatic processes which come with age. In the first place *symptoms and signs of disease are much less conspicuous than in younger patients*. As most of the clinical phenomena indicative of disease, such as fever, pain, tachycardia, swelling and the like are manifestations of the reactions of the organism to injury, it is not difficult to appreciate why this should be so. In older people these reactions are less violent and thus illness is relatively asymptomatic. This is conspicuously manifest in connection with appendicitis in the aged (see Chapter 34), tuberculosis (see Chapter 22), pneumonia (see Chapter 23), and diabetes mellitus (see Chapter 14). Minor deviations and subtle suggestions of dysfunction take on great significance.

Secondly, *repair is slowed*. Fractures heal more slowly (see Chapter 43), wound healing is likewise retarded.⁷ Less obvious repair, such as that required after acute infections, after fatigue, after intoxications and the like, is also slowed. Therefore more time is required for convalescence. For practical clinical purposes, it is feasible to assume that for each five years of age an additional twenty-four hours is required for postinfective repair. Whereas a child of five will recover from the effects of an acute infection in twenty-four hours, a man of sixty will require twelve days. However, because the man of sixty did not have a violent fever or feel very ill (lessened reaction) it is difficult to convince him of the need of such a long convalescence. Frequently it is wise to compromise for one day extra for each ten years of life. Prolonging convalescence does not imply complete immobilization. Long immobilization is not desirable for the elderly (see also Chapter 8). By making the increase in activity very gradual, permitting a little more exertion each day, we strike the happy medium between unnecessary bed rest and time enough for repair before resumption of full duties.

The third clinical implication of slowed homeostasis is also of therapeutic significance. The older patient has a *narrowed margin of safety* in the poor tolerance to stresses. The effects of an impairment of function are more serious, compensatory activities by other physiologic processes are weakened

volume of blood flow. When the vascular lumen is narrowed, elongated and tortuous, the *quality* of the blood reaching the capillaries takes on increasing

importance in proportion to the impairment of flow. Aging parenchymatous cells are less tolerant to histanoxia.⁸ Similarly the cardiac overload of obesity is better carried by a young myocardium. Treatment of the aging and the aged must neglect no detail in trying to keep weight, hemoglobin content, blood protein, blood sugar, and the like at optimum levels. (See Chapter 6.)

Reactions to Drugs. Several factors have to be considered here. Orally administered drugs are absorbed more slowly and sometimes less completely. Urinary elimination is likewise retarded and diminished. Thus drugs, as foreign substances, are more likely to accumulate, especially if circulatory impairment be added to the effects of renal aging.

Several substances are known to be more toxic to older individuals than to younger persons. All the barbiturate sedatives are poorly tolerated. They frequently enhance disorientation, forgetfulness, apprehension, and unsteadiness. The senile are often so confused by barbiturates that they become acutely anxious. Bromides and potassium thiocyanate, too often prescribed indiscriminately in hypertensive disease, tend to be cumulative in effect and may lead to serious intoxications. Bromism is often associated with excitation, which will only be further aggravated by more bromide ions. Ouabain is less well tolerated by older experimental animals, the studies in relation to digitalis are indecisive in regard to tolerance change with age. Nevertheless White⁹ advises smaller doses of digitalis for old people.

Tolerance for insulin, histamine, and the opiates is somewhat increased with age. Nitrites are much less active pharmacologically after the peak of maturity.¹⁰ Experiments indicate that in experimental animals the tolerance for thyroxin diminishes after maturity,¹¹ but this does not coincide with our own observations on man. Tolerance for tobacco, caffeine, and alcohol varies widely. Individual variation appears to be independent of age. Alcohol in moderation is an extremely useful adjunct in the management of the aged. It relaxes tensions, induces a sense of well being, and may stimulate appetite.

Nutritional Variation. Dietary habits play an important part in determining the nutritional status of elderly people. Fixation of habits is characteristic of aging, though not solely because of personality fixation which is the usual accusation made against the aged. Habits arise and become fixed by repetition over a period of time, the longer the repetition is continued the more rigid the fixation. Fads and fancies may contribute to some habitual dietary peculiarities, but they are less significant than the fact that most people avoid the foods that they have learned are not well digested. Likes and dislikes more frequently than not parallel tolerances and intolerances. Therefore the dietary habits of the elderly should not be brushed aside arbitrarily as being due to prejudice. Nor should we assume that they are necessarily undesirable.

minerals (especially calcium and iron), vitamins, and protein is not uncommon. (See Chapter 12.) When dentures are lacking or ill fitting, the selection

of the milder late type of diabetes are avoidable if the dietary is kept better balanced in senescence (See Chapter 14) Older people living alone and lacking interest in eating and therefore devoid of enthusiasm for cooking will select chiefly packaged pre prepared foods such as bakery goods Because the senile bowel cannot handle roughage, patients frequently avoid almost all fruit and vegetables and thus fail to obtain adequate soft bulk Constipation is frequently due to a failure to comprehend the necessity of soft bulk and liberal fluids (See Chapter 34)

TABLE 2
PROPER WEIGHTS FOR MEN
(Allowing Plus or Minus 5 Pounds)

Height in Inches	Weight in Pounds		
	Small Frame	Medium Frame	Large Frame
62	120	128	136
63	123	131	138
64	127	135	143
65	131	139	147
66	134	142	151
67	138	146	155
68	141	150	159
69	146	155	163
70	150	160	167
71	155	163	172
72	158	167	177
73	163	172	182
74	169	177	187
75	174	183	193

Anorexia is often psychogenic, especially when the "will to live" and enthusiasm for accomplishment are weakened by long disabling illness Small frequent feedings are often much better tolerated than larger meals Where the appetite is poor, it is important that the food prescribed be easy to eat Patients are not interested enough to make an effort to eat, nourishing beverages should be encouraged¹²

During senescence obesity from sheer overeating is a common and serious problem Obesity becomes increasingly detrimental with advancing age Par

importance in proportion to the impairment of flow. Aging parenchymatous cells are less tolerant to histanoxia.⁸ Similarly the cardiac overload of obesity is better carried by a young myocardium. Treatment of the aging and the aged must neglect no detail in trying to keep weight, hemoglobin content, blood protein, blood sugar, and the like at optimum levels. (See Chapter 6.)

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The eating habits of older patients are affected by many factors. Appetite is variable. In true senility it is often greatly depressed, roughly paralleling the gastric achlorhydria. As a result, undernutrition due to diets deficient in minerals (especially calcium and iron), vitamins, and protein is not uncommon. (See Chapter 12.) When dentures are lacking or ill fitting, the selection of soft and readily masticated foods in preference to bulkier and firmer items

minder, a glass placed where it gets in the way will suffice. It just doesn't occur to the busy executive to drink water, he must be reminded of the need until a new habit pattern is developed. Then it will no longer be necessary. The element of habit in any nutritional problem, be it individual or *en masse* as in families, institutions, or communities, must never be ignored. Habits must not be abruptly violated, but gradually modified.

Psychologic Factors Aging brings about mental changes as well as physical alterations. Elderly persons differ *both intellectually and emotionally* from what they were in early and middle maturity. These changes in psyche are insidious and continuous, just as the subtle biochemical and structural alterations of the soma. There are no sharp lines of demarcation defining intellectual and emotional maturity (see Chapter 5). Here, as in somatic health, vigor, or biologic age, values are wholly relative. There is manifest, however, even more asymmetry and individual variation in psychologic maturation, and later involution, than in the organic changes consequent to senescence. Therefore generalizations are apt to be misleading. Nevertheless, because the psychologic changes follow certain basic patterns, or thematic forms, we are justified in describing a composite picture. These fundamental trends, though they may be expressed differently in different individuals and vary considerably in intensity and in age of insidious onset, are most significant to accurate diagnosis and wise therapy in geriatric medicine.

The alterations of personality which occur with normal aging are quantitative rather than qualitative. Notable changes in characteristics and emotional reaction patterns strongly suggest mental illness. The personality characteristics developed in youth and early maturity, however, become considerably intensified and fixed. As certain traits may have been fairly well concealed or not observed in youth, there may be some apparent change, but analysis of past behavior almost invariably reveals the same personality existing throughout life. The leopard does not change his spots with age; they merely become blacker and therefore more conspicuous. Thus the *intrinsically* anxious, economically insecure and parsimonious middle-aged man will become the grasping miser; the sanctimonious egoist becomes an intolerably opinionated bigot; the querulous complainer or clinging vine becomes hypochondriacal and increasingly helpless and demanding of constant attention; the generous more generous and the tolerant even more liberal and understanding. During youth and early maturity the necessity for herd conformation frequently leads to partial repression of the overt evidences of such personality characteristics; in later maturity and particularly in senility, the necessity of conventionality becomes less and these traits become more conspicuous. It is highly questionable whether there actually is such a thing as a 'second childhood'; those who become conspicuously childish in their attitudes and behavior in their dotage are merely continuing their first childhood. They never did mature intellectually or emotionally. Immaturity can be fairly well concealed from all but trained and searching eyes by strict adherence to conventionality. Emotional maturation is frequently greatly retarded and thus psychologic asymmetries are more conspicuous in later years.

As stated above, age brings about fixation of *habits*. This applies to intellectual and emotional habit patterns as well as to the somatic and automatic habits of bowels and the like. However, it is unjust and illogical to attribute rigidity of habits solely to aging. The mode of life has a good deal to

habit formation. A life of repetitive routine, limited in scope and interest, can but lead to further narrowing and rigidity. Breadth of view and catholic interests are hardly to be expected of one who for twenty or thirty years has devoted himself or herself to purely routine tasks such as accounting, selling shoes, or keeping house. Flexibility, like any other functional capacity, atrophies with disuse. The only fundamental difference between a rut and a grave lies in the length of the former. The aging and the aged who have lived with varied interests, diverse activities, and broader perspective do not reveal much, if any, of the fixation or mental rigidity so generally observed in the senile. However, in all older people, the fact that years of repetition tend to make habits deeply ingrained must be considered in outlining any regime.

Memory, especially for recent events, becomes less reliable with age (See Chapters 5 and 18.) Again one is not warranted in assuming that all the depreciation in memory is due to depreciation of functional capacity to remember. The will to remember is lessened, motivation is less urgent. With changing values, petty details become less significant and impressions therefore become less brilliant.¹³ Disinterest and inattention play significant roles in creating defects in memory, preoccupation with other matters of greater interest is also a factor. The older man has little interest in names and telephone numbers. And lastly, loss of acuity of the special senses blurs the original impressions received and thus makes remembering of poorly observed details impossible. It is not easy to recognize a face seen only through the haze of a cloudy lens or to identify a voice heard but poorly.

However, irrespective of the mechanisms involved in the pathogenesis of memory impairment, the phenomenon itself is extremely significant to effective application of geriatric medicine. In the first place, we must learn to be very skeptical regarding the accuracy and completeness of the past history as given by an aged patient. Innocent lying by omission of forgotten illnesses is common and can be misleading unless leading questions and several repeated efforts at eliciting the story of the past are applied. Patience, thoroughness, and repetition are necessary in taking the history of older patients if we wish to be reasonably certain of obtaining all pertinent information. (See Chapter 7.) Secondly, therapeutic instructions to elderly patients must be precisely specific, and preferably written or stated in the presence of a nurse or younger member of the family. The aged cannot be expected to remember the details of half dozen or more therapeutic suggestions given verbally at times when they are excited and a little confused by unusual visits to their physician.

their physical dependency, senile patients are apt to become emotionally dependent upon their physicians. Sometimes such dependency is very helpful in building morale, sometimes it must be curbed and restrained else the patient become increasingly incapable of even partial self sufficiency. It is notable that senescent or mature patients have learned not to expect miracles, they are more deeply and warmly grateful for assistance, kindness, and patience than any other group.

upon the younger generation. A survey made in New York State in 1929, and therefore before the great depression, revealed that of people aged sixty-five, only 5 per cent were able to continue at near their previous standard of living on accumulated savings, that 28 per cent continued employment for support, and that 67 per cent were dependent upon either family or state.¹³ At present and in the near future opportunities for saving for old age are even less, heavy taxation makes accumulation of money extremely difficult. Social Security old age benefits of perhaps fifty dollars per month hardly touch the problem except for those previously in the very lowest income brackets. The anxiety which is so often felt but rarely expressed verbally thus has foundation in reality.

The supreme tragedy of senility is awareness of uselessness. The truly aged are *not* needed by their families or by society. Whether the rejection from useful occupation is warranted by infirmity or not is immaterial to the individual. He needs to be needed by those close to him and by the community. A purposeless existence predisposes to psychologic imbalance and is frequently a factor in mental breakdowns. One of the most potent and effective therapeutic implements is to give *purpose in life*. Frequently, the intellectually competent old person is well aware of the fact that his family is merely waiting for him to die, no matter how hard the younger generation attempts to conceal the fact. This awareness is a significant etiologic factor in the development of the violent paranoid antagonisms (typically directed against those superficially nearest and dearest) characteristic of arteriosclerotic dementia.^{12, 14} (See Chapters 11 and 17.) Insistence upon continuation of useful occupation even in the face of somatic reasons for caution against effort may retard psychologic decompensation for many years. There is greater hazard for the aged in being too restricted in their activities than in their overdoing.¹⁵

The emotional problems of the climacteric are discussed elsewhere.¹³ (See Chapter 41.)

The borderlines between mental health and mental disease are as indefinite and broad as the relativity of somatic health. Awareness of the changes and emotional problems of aging as discussed above on the part of the physician can do much to make geriatric medicine more effective. Receipt of advice without the motivation to spark the effort necessary to put it into effect is as useless as filling the gasoline tank of an automobile with an exhausted battery. It is part of the function of geriatric medicine to rekindle, if possible, the *desire* to carry on and to accomplish.

Basic Clinical Implications. Aging brings about many and varied changes in soma and psyche. These changes create certain functional characteristics peculiar to the biologic age of the individual. The older person is a different person from what he was in the past. We have discussed some of the more significant and typical of these differences. Their practical clinical implications can be summarized very briefly.

Individualization of the mature and old patient is of paramount importance. Individual variation increases with age. This applies with equal force in diagnosis and in therapy.

The *symptoms and signs* of functional depreciations are subtle and insidious. Waiting for obvious or conspicuous evidence of disease means waiting too long. Homeostatic mechanisms are weakened by age, the body's reactions to injuries are less violent.

The *functional capacities or reserves* are most significant. Early depreciations can be discovered only by studies under conditions of stress. The outlook in an acute illness is greatly affected by the vitality or degree of health of the patient prior to the acute disorder. The values of a base line, determined by health inventory, are far more than merely reassuring. Health is always relative.

The *individual is indivisible* and must be treated as such. Psyche and soma cannot be separated. morale, confidence, patience, understanding, and recognition of habit patterns are as significant as pills, lotions, surgery, vaccines, splints, or hydrotherapy. Maturing is not always easy. Involution can be modified and retarded by wise guidance. The concern of geriatric medicine is not with disease alone. It is primarily interested in the aging and the aged *individual*. The patient is more important than his illnesses. Therapy must therefore consider all the multiple facets of change introduced by senescence.

CHARACTERISTICS OF THE DISEASES COMMON IN SENESCENCE

There are few, if any, diseases which may not occur at any age. The aging and the aged are not wholly immune to any disorder. The acute communicable diseases of childhood are not unusual late in life. During one winter season we had occasion to see eight cases of parotitis in grandmothers, the mothers carried sufficient immunity acquired in childhood to protect them, but this had disappeared in the grandmothers. Pertussis, scarlet fever, and measles can be very serious illnesses in the aged. The potentiality of an epidemic in institutions for the aged must be kept in mind.

However, after maturity the acute infective disorders become less frequent and a group of so called degenerative disorders becomes increasingly common. These are chronic, slowly progressive disorders. They constitute the gravest menace to continued health and usefulness of those reaching maturity.⁴ Today nearly 60 per cent of all deaths in the United States are attributable to chronic diseases. The frequency of the degenerative disorders is increasing, perhaps largely because so many more people survive through youth to fall into the vulnerable age period after forty.¹³ The social significance of chronic progressive disease is discussed in the preceding chapter.

Four major groups of these disorders are particularly significant to geriatric medicine:

- 1 Circulatory impairments
- 2 Metabolic dysfunctions
- 3 The arthritides
- 4 Neoplasms

It is obviously impossible to discuss these many and complex disorders adequately in a single chapter, they are to be considered fully elsewhere in the text. At this point we are concerned with basic principles and therefore with certain characteristics common to a category of disorders. The circulatory and metabolic groups are the most important from the viewpoints of *frequency*, *disablement*, and *mortality*.⁴ (See Chapter 1). Included in these two groups are

- 1 Circulatory Disorders
 - 1 1 Hypertensive arterial disease
 - 1 2 Arteriosclerosis
 - 1 21 Cerebral

- 1 22 Coronary
- 1 23 Renal
- 1 24 Aortic
- 1 25 Retinal
- 1 26 Pancreatic
- 1 27 Extremities
- 1 3 Primary cardiac disease
 - 1 31 Valvular disease
 - 1 32 Myocardial disease
 - 1 33 Pericardial disease
- 1 4 Anemias
- 2 Metabolic Disorders
 - 2 1 Anemias
 - 2 2 Diabetes mellitus
 - 2 3 Thyroid imbalance
 - 2 4 Gout
 - 2 5 Climacteric (female and male)
 - 2 6 Renal decompensation

It is obvious that several of these disorders overlap and that arbitrary classification is impossible. Perhaps the best way to clarify the significant generic characteristics is to contrast these abnormalities to the illness frequent in youth.¹⁶

Youth		Senescence	
Etiology	Exogenous Obvious Specific (single) Recent	Etiology	Endogenous Occult Cumulative Multiple (superimposed) Often past
Onset	Florid	Onset	Insidious asymptomatic
Course	Acute Self limited Immunizing	Course	Chronic Progressive (long disability prior to death) Not protective (increase vulnerability to other diseases)
Little individual variation		Great individual variation	

Etiology The chronic progressive disorders so significant in later maturity all arise from *multiple* and *variable* etiologic factors. The causative influences are *accumulative* and *obscure*.¹⁷ In no two instances are the etiologic factors necessarily identical. This situation is in sharp contrast to the etiologic patterns of the acute exogenous infective disorders of youth. Medical thinking is still suffering from the retarding influence of over enthusiastic acceptance of the concept of specific etiology which came into being with the birth of bacteriology. Koch insisted that etiologic relationship was not established unless (1) the microorganism is present and demonstrable in every case of the disease (2) it is cultivated in pure culture (3) inoculation from such culture reproduces the disease in susceptible animals and (4) it can be reobtained

from such animals and again grown in pure culture. These postulates became the basic law of the new science, bacteriology. Feverish research, spurred by the hope that in this vast realm of pathogenic bacteria would be found the explanation of *all* disease, led to many brilliant and invaluable discoveries. One after another, the specific organisms of infective diseases were identified, isolated, and studied. But the dreams and hopes of the earlier bacteriologists have not materialized. Invading microorganisms do not cause all disease, and even when they are involved, they are not the sole cause of infective diseases. The concept of specificity, though invaluable in preventing many hasty and erroneous conclusions, also blinded many scientists to the fact that disease follows only when the germ invades a *vulnerable* host.

Causative influences are amenable to analysis. Such analysis reveals three categories of causative factors. Breakdown of etiology into these three types of factors has proven extremely helpful in understanding the ways by which disease comes about.

- 1 Predisposing factors
- 2 Provoking factors
- 3 Perpetuating factors

Application of this analytical tool is particularly pertinent to the problems of the causation of the degenerative disorders. As both curative and preventive therapy must be based upon correction of etiology to be truly effective, the obscurity of causation of these disorders constitutes our greatest handicap to successful treatment. Unremitting *search* and *research* for contributing factors is requisite to therapeutic success, especially early in the course of these progressive illnesses. It may be said that failure to control the progression of hypertensive disease, for example, implies failure to elicit and correct all the etiologic factors.

Silent Onsets. All these disorders begin silently and asymptotically. They exist, frequently, for years, before they are grossly detectable. Though the incidence of *discovered* cases of vascular diseases rises sharply after forty years of age, the changes started years earlier. These are fifth column diseases, silent saboteurs. To be discovered early, they must be searched for. (See Chapter 6.) Functional impairment is never obvious until it reaches a degree incompatible with maintaining homeostasis. (See Chapter 37.) Early depreciations therefore are detectable only by the application of stress under controlled conditions.

Furthermore, what symptoms and signs do arise are secondary and indirect. For example, there is no kinesthetic sense which informs us of elevation of the arterial tension. Impairment of the circulation from whatever cause (hypertension, sclerotic narrowing, anemia, cardiac decompensation) will produce symptoms of functional impairment of the parenchyma through oxygen starvation. Cerebral malcirculation results in neurologic and psychiatric symptoms, only indirectly associated with the vascular system. Impair-

variably several disorders are superimposed. Mixed forms are the rule rather than the exception. In anatomic and functional diagnosis we must guard against oversimplification just as much as in considering etiology (See p. 40). Too precise definition of clinical entities is a vice rather than a virtue in geriatric medicine. Such disorders as diabetes mellitus, arteriosclerosis, hypertrophic arthritis, and/or hypertensive disease are often thought of as sharply demarcated specific entities, whereas actually they are not so separable.¹⁸ For example, in hypertensive arterial disease we must assume simultaneous cardiac injury even though it may not be conspicuously manifest. Similarly, arteriosclerosis with predominant cardiac (coronary) symptoms probably does not exist without less obvious but potentially significant cerebral and renal arteriosclerotic change. A coronary occlusion differs from a cerebral thrombosis only in location of the dramatic, acute phenomena. Hypertrophic arthritis in the aged is frequently associated with superimposed gout, therapeutically both disorders warrant attention. And to cite a last but important example, it should be emphasized that anemia is more often than not of mixed pathogenesis, attributable to both primary hematopoietic inadequacy and nutritional deficiency.

None of the chronic progressive disorders of later years confers immunity. Rather do they increase the vulnerability to related involutional or degenerative depreciations. Frequently several elements in their pathogenesis contribute to the causation of other difficulties. A conspicuous illustration of such interdependence is found in hypertensive disease, well known to be of most varied etiologic background (see Chapter 30). Hypertension is frequently associated with anemia; correction of the anemia reduces the blood pressure to more nearly normal levels, hypertension impairs the renal circulation and efficiency, renal impairment is a significant factor in the causation of stubborn anemia. Similar vicious circles link others of these disorders and contribute to their characteristic progression.

Progressiveness. The intrinsic tendency to slow but persistent progression is a generic characteristic of great importance. None of these disorders is self-limited, there is no tendency toward spontaneous cure. Though remissions do occur, they are usually brief and progressive deterioration is soon resumed. Progression is inherent in the pathogenesis of many of these disorders; it is enhanced by associated difficulties. For example, obesity certainly contributes to progression of hypertrophic arthritis of the weight-bearing joints such as the knee or hip.

Progression is usually slow. Thus long periods of slowly increasing disability precede the final outcome in death. The protracted disablement of arthritis is not revealed by mortality statistics.⁴ The leisurely pace of progression is at the same time an asset and a liability to the physician dealing with older patients. It gives him time for the thorough and comprehensive analysis required for effective therapy. But it also implies that therapy to be truly effective must be instituted very early in the course of these disorders, before they become irrevocable. The most effective time is *before* the appearance of obvious symptoms and signs. At this time, unfortunately, the patient is usually not aware of his progressive illness, feels too well to be alarmed, and does not appreciate the problems of the future. Thus the physician is greatly handicapped in his efforts at prevention and retardation. It is in prophylactic, constructive medicine that geriatric medicine has its greatest potentialities.

Though *cure* of the chronic progressive disorders of later years is often impossible in the conventional sense, *control* is feasible and frequently extraordinarily effective.¹⁹ Therapy to control is perhaps best illustrated by the management of diabetes mellitus. The diabetic, on a properly controlled diet with or without insulin, is in physiologic balance and for practical purposes essentially well, though he remains a diabetic. The disease is not cured, in the sense that it is over with and can be forgotten, it is, however, controlled. A similar situation applies today to pernicious anemia, nutritional hypochromic anemia, hypothyroidism, gout, climacteric disturbances, and the like. To a lesser degree, control therapy can and does retard the progression of hypertensive arterial disease, arthritis, and arteriosclerosis. Frequently maintenance of the patient in a *status quo* is as much of a therapeutic accomplishment as one can expect. It is often wisest to explain to the patient the irrevocable nature of the lesions of his disorder lest he continue to search for impossible miracles.¹³

Physiologic Consequences. The primary pathologic changes of most of the chronic and progressive circulatory and metabolic disorders arise in supporting structures or the matrix tissue.²⁰ Nevertheless the critical injury is to parenchymal cells. The damage is done by interference with cellular nutrition. A broad concept of nutrition must include not merely the ingestion of nutrients, but their effective absorption and transportation. Furthermore, oxygen is just as much a nutriment as glucose and equally necessary, water is as vital as any vitamin. The vascular system is concerned primarily with food transport and the removal of metabolic debris.

These disorders impair parenchymal nutrition by any one or more of the following mechanisms:¹⁶

- 1 Inadequate supply
- 2 Ineffective transport
- 3 Inefficient utilization
- 4 Insufficient removal of metabolic debris

These factors are intimately interrelated and frequently overlap. For example, histanoxia, or cellular oxygen want, may be induced by a combination of lowered oxygen-carrying capacity (anemia, perhaps of nutritional deficiency origin), arteriolar constriction and capillary stasis as in hypertension, and partial cardiac incompetence. The net result is asphyxia of parenchymal elements. Perhaps, because of hypoinsulinism, glucose combustion is further impaired. All these factors may coexist.

Identical consequences follow the apparently normal changes of aging *per se*, but much more slowly. It may be justifiable to classify many of these chronic and progressive deteriorations as pathologically accelerated and asymmetric senescence. This concept is at least open to argument. It is im-

Such emphasis must not permit us to be misled into thinking that all these

problems have but a single solution, and because of the several common denominators they can be considered as one. Individual variation in the diseases of later years is as significant as the variations in the patients having the disorders.

are usually parallel, but not always. The rate of progression is a most significant element in evaluating prognosis.

We are still unable to explain why arteriosclerosis in one individual affects the coronary vessels more than other arteries, or why in another the cerebral vessels appear to give way first. Whether the difference lies in the disorder or in selective vulnerability of the individual is an open question. The great variations in distribution and intensity of the lesions of arteriosclerosis accentuate the importance of individualization in diagnostic study and therapy of senescent individuals. Variability undoubtedly depends in some way on the fact that each individual has been subject to a different series of insults and injuries in different sequence. The cumulative effects of the innumerable intoxications, transient infections, psychic and physical traumata which constitute the vicissitudes of our existence probably play a major role in determining the vulnerability of this or that structure. Again we must point out the importance of treating the patient and not merely his disease.

SUMMARY

Geriatric medicine, dealing as it does with the care of the aging and the aged, must take cognizance of all the structural, physiologic, and psychologic changes which result from aging. To age is to change. Geriatric medicine is

environment which is affected by their own aging and by the aging of many others. Geriatric medicine is an art as well as a science.

Aging may be normal or abnormal. The line of demarcation is not sharp, for health is relative. The relativity of health is nowhere more conspicuously manifest than in the aging. Age is asymmetric, health likewise varies within the individual. The changes of age arise from both the passage of time in living and the accumulation of injuries inevitable in an active life. These changes affect clinical practice in all its phases: diagnosis, prognosis, and therapy. Let it be remembered that diagnosis exists primarily for the purpose of guiding therapy and therefore must include consideration of the patient's preexisting depreciations, individual idiosyncrasies, and functional capacities. The individual is indivisible.

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CHAPTER 3

PHYSIOLOGIC CHANGES OF NORMAL AGING

ANTON J. CARLSON

INTRODUCTION

THE sciences of biology and medicine have not yet reached the stage when a comprehensive factual chapter on the physiology of aging can be written. At the end of an excellent fifty-page review of the chemical aspects of aging, McCay¹ concluded that "almost nothing is known of the biochemistry of aging." The physiologist need not be quite so pessimistic, but instead of writing on what we do not know, biochemists and physiologists might be better employed in research on these important unknowns.

Many age changes in the animal body have been clearly established. The cause or causes of these age changes in structure and function are still obscure. We are not yet in position to determine with the certainty of science to what degree the changes in the machinery of the body which parallel advancing age are due to the genetic constitution of the individual and the species, that is, to the aging process, *per se*, and how much they are due to the accidents of living, such as faulty diets, infections, overwork, laziness, or gluttony. As regards the life span of the species, plant or animal, undoubtedly the genetic constitution plays the primary role, for on no other basis can we account for some species of plants living only one year or less and other species living several thousand years. Disease and other accidents of living cannot account for such great differences in life span in plants, nor can it account, in my judgment, for similar great differences in the life span in different species of animals.

There is growing evidence that *hereditary constitution* is also a factor in the life span of specific individuals within the species, irrespective of ignorance, faulty diets, infections, and other accidents in the life of the individual. This appears also to be true for the life span of individual organs or systems within the individual. For while it is true that faulty diets, infections, etc., may hasten the atrophy of the ovaries in primates and thus bring on a premature menopause, and that similar accidents and diseases may hasten the loss of elasticity of the lens of the eye, it is nevertheless true that the life span of these two organs, the primate ovaries and the lens of the eye, runs on the whole such a fairly regular course in the individuals of the species that the early atrophy of the ovaries in the female and the relatively early decrease in and final loss of elasticity of the lens can hardly be accounted for by the accidents and stresses in the life of the individual. The life span of these two organ systems must be primarily determined by a physiologic time clock in particular genes of the hereditary constitution. But when we consider the other organ systems of the body, the slowing up of function, or the impairment of function, is on the whole so gradual, at least in all cases where the life of the individual is not specifically terminated by overwhelming accidents in individual organs (such as malignant growths, gastric ulcer, pneumonia, tuberculosis,

nephritis, or arteriosclerosis) that it becomes practically impossible to separate the factors of aging from the factors of impairment due to the accidents of disease. Of course the germ cells themselves may be acted on unfavorably by

of the species, we would have degeneration of species and shortening of the life span of the species, and this would be a rule instead of the exception

The relative importance of the life span and of the genetic constitution in

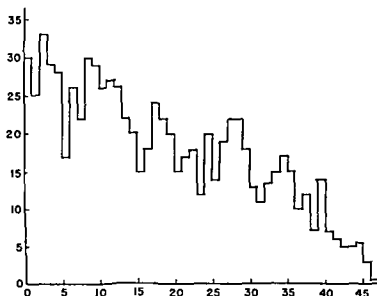


Fig. 10 Graph of the fission rate in successive ten day periods in isolation cultures of *Pleurotricha lanceolata*, in the experiments of Baitzell, 1914. The horizontal scale shows the number of successive ten day periods, the vertical scale the number of fissions in each period. The fission rate declines (with many fluctuations) till the culture ends in death (Baitzell J. Exper. Zool., 16)

most extensively modified genetic constitution leading to the earliest senescence. To be sure, it is a long distance from the fruit fly to man, and by and large science has yet been able only to degrade rather than to strengthen the genetic constitution by experimental means. Nevertheless, similar factors probably play a role in the phenomena dimly foreshadowed in the life of man, that is, in the relatively long lived and relatively short lived human families.

If further justification of our primary thesis, namely the practical impossibility of separating the factors of aging *per se* from the effects of the accidents of living, is needed, we might cite the case of many plants in which tissue de-

age degeneration seems to be due to progressive chemical changes in the cell nuclei, changes which in turn interfere with later cell division and growth.

Unicellular animals are commonly said to be immortal. Nevertheless, these lowly cousins do seem to degenerate with age. Again, this degeneration appears to be in the physiologically active nucleus, and unless this aged nuclear tissue is replaced by the reserve micronucleus, as occurs in conjugation and endomixis, decline and death of these unicellular organisms seem inevitable. This is, in principle, not so different from sexual reproduction and death of the soma or body in the higher plants and animals.

The debate on heredity vs. the strain and the accidents of living as the cause or causes of aging is from now on a waste of time and paper. It is not an either/or. Evidence now at hand shows conclusively that both play a role in the aging process of man and animals. If longer life or longer abundant life is the desired goal, biologically and socially, we must obviously reduce the production of those with less than optimum hereditary potentials, and provide optimum diets and an optimum environment for the others. This includes less disease and fewer maiming accidents.

of the individual. The universality of these age changes, even in individual persons whose hereditary constitutions have been able to meet the accidents of living for one hundred or more years, renders it highly probable that we are here dealing with age changes primarily inherent in the constitution of living matter, no matter how greatly these changes may be speeded up by the accidents of living.

Progressive age changes, not as yet shown to be due to specific diseases are

1. Gradual tissue desiccation. By determining the electrolyte concentrations in the tissue cells some doubt has been raised as to the reliability of the older experiments which seem to establish a gradual tissue desiccation as part of the aging process.
2. Gradual retardation of cell division, capacity of cell growth, and tissue repair, including reduced capacity to produce immune bodies in case of infections.
3. Gradual retardation in the rate of tissue oxidation (lowering of the basal metabolic rate).
4. Cellular atrophy, degeneration, increased cell pigmentation, and fatty infiltration.
5. Gradual decrease in tissue elasticity, and degenerative changes in the elastic connective tissue.
6. Decreased speed, strength, and endurance of skeletal neuromuscular reactions.
7. Decreased strength of skeletal muscle.
8. Progressive degeneration and atrophy of the nervous system, impaired vision, hearing, attention, memory, and mental endurance.
9. Gradual impairment of the factors which in the normal person in his earlier years maintain a fairly constant internal environment for the cells and tissues (homeostasis). It is evident that sufficient

weakening of any one of the numerous essential links in the complex process of homeostasis will produce deterioration ²

These changes are not listed in the order of their primary significance, for this is not yet known. Nor does the list imply that these are all the changes of senescence. It is already evident that aging is a continuous and complex process, some changes starting early, others later in the life of the individual.

This picture of aging is, in fact, not so dark and depressing as it seems, for the following reasons: (1) An efficient mental life is possible, even with reduced factors of safety. (2) Moderation in all things, plus competent medical guidance in regard to the accidents of disease, will tend to prevent undue corrosion of specific life links, so that all of them pass down the hill gradually and in step. (3) The man or woman who has passed the first sixty or seventy years in honest toil and persistent efforts at understanding has or should have accumulated a *great reserve of wisdom*, now at the disposal of the younger generation, a reserve of wisdom which antidotes the stresses and strains induced by the fears, the vanities, the greeds, and the ignorance of the earlier years. This is true at least up to the point where the impairment of cerebral function leads to the so-called "second childhood" (See p. 265). Intensive research on the many unknowns in the aging processes will some day enable the science of medicine to delay greatly this involution. This is not "rejuvenation" either in the popular or in the quack medical sense. *It is merely better care and better driving of the living machine, provided by our growing understanding. This is the goal of geriatric medicine.*

THE DIGESTIVE SYSTEM

The salivary glands usually show some evidence of atrophy with advancing years, competent investigators have reported definite decrease in *salivary volume* and in percentage of ptyalin with advance in age. This is not a vital link in the life span, for most animals have no ptyalin and we can adequately digest all our food without the aid of ptyalin, the most important constituent of saliva being the water and mucin. To what extent the decrease in saliva or the age changes in the composition of the saliva permit or favor growth of the aciduric flora of the mouth, which in turn may enhance degenerations in teeth and gums, is still an open question.

The work of Pollard indicates that from the age of twenty years on, the *volume and the acidity of human gastric juice* decrease definitely but gradually and it is well established that the incidence of achlorhydria increases with age.³ Thus achlorhydria appears in only about 5 per cent of people under the age of twenty years, while it appears in 35 per cent of the population above the age of sixty. Again, we must note that the higher incidence of achlorhydria with age may be due to the accidents of living rather than to the process of aging. Nevertheless, the existence of achlorhydria in a child of five and the presence of hydrochloric acid in gastric juice of a person at the age of one hundred

or intrinsic factor of red cell formation. The gastric pepsin factor seems less

subject to decline with aging and to the accidents of living than does the hydrochloric acid factor.

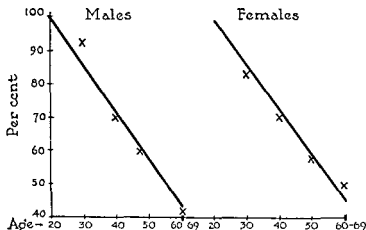


Fig 11 Percentage decrease in total output of gastric juice acid (volume times acidity) with age in normal persons. Note that when the volume of secretion is considered, the total acid secreted decreases with age similarly in both sexes. When acidity alone is considered, a decrease appears to occur only in the male (Pollard, Arch Int Med, 16)³

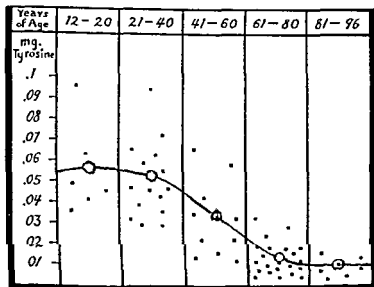


Fig 12 Pepsin in gastric juice. The concentration falls sharply during the seventh decade of life and after that maintains a rather constant low value (Meyer, Spier and Neuwelt, Arch Int Med, 65)⁴

In connection with the *motor functions of the stomach*, in the absence of such diseases as cancer or ulcer, there is little clear evidence except that dealing with the strength and rate of the contractions of the empty stomach. that is.

the so-called hunger contractions. These do definitely decrease with age, as shown particularly in man and dog. The periods of relative motor quiescence of the empty stomach are shorter in the young, become much longer in advancing years, and the periods of active contractions shorten, and the contractions themselves become feebler with advancing years. But this evidence of impairment of gastric motor activity with aging appears to be insufficient to cause significant delay of the emptying time of the stomach, and not significant enough to produce impairment of digestion.

Atrophic gastritis might readily be a chronic effect of dietary indiscretions, if not of chronic infections, wherein such processes wear out, as it were, the regenerative or recuperative capacities of the gastrointestinal mucosa. However, there is no good evidence of any gradual increase of atrophic gastritis with age.

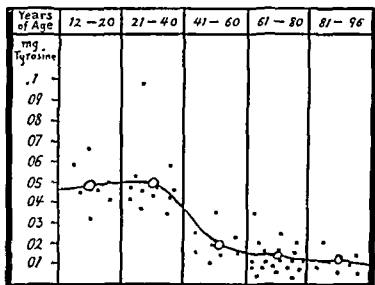


Fig. 13. Trypsin. The tryptic activity falls sharply after the fortieth year of life. The proteolytic powers of pancreatic juice bear no relation to gastric acidity (Meyer, Spier, and Neuwelt Arch. Int. Med., 65).

In the case of the pancreas, the incidence of *diabetes* increases with advancing years, at least up to fifty or sixty. The pancreas of old people shows many scars from accidents and injury of living. Goodpasture has described degenerative changes in the pancreas of old dogs and similar changes have

been indicated by pancreatitis, liver, or biliary tract disease. The fact that in some individuals diabetes may develop in early childhood, while in others the hormone mechanism of the pancreas is able to meet the metabolic need of an individual one hundred years old, would seem to indicate that the failure of this phase of pancreas function with increasing age is due to hereditary weakness rather than to the accidents of living.

While the gross weight of the liver decreases with age, at least from the fourth to the seventh decade, the present available liver function tests do not reveal any *liver impairment* with aging in the absence of specific liver disease. In fact, the livers of old dogs appear to be more resistant to the injurious effects of chloroform than are the livers of pups. This is also clearly the case in the human species. But it is well known that the incidence of liver pathology increases with age and there appears to be some decrease in concentration of vitamin C in the liver with advancing years. Whether or not this is secondary to decreased absorption of this vitamin from the intestine is not known. The incidence of gallstones increases with age, but the gallbladder, free from disease, apparently empties normally even in very old people and very old animals.

In comparison with "normal" persons at forty years of age and below, a percentage of such "normal" persons at sixty-five years of age and above shows a reduced capacity to restore the blood sugar to its average level after ingesting a quantity of glucose. The precise impairment (liver, pancreas, skeletal muscles) responsible for this retardation is not yet clear. The same retardation in the restoration of the acid-base balance after ingestion of 25 gm. of sodium bicarbonate has been reported in aging individuals.

There is some evidence of decreased ability of the aging body to handle cholesterol. Whether this impairment is due partly or entirely to the less efficient liver is still an open question.

There appear to be no reliable data on atrophic changes in the mucosa and in the musculature of the *small intestine* with advancing age. To be sure, death from intestinal obstruction increases with advanced age, but this may be due to adhesions of pathologic origin rather than to weakness in the intestinal musculature. Atrophy of the colon musculature with advanced age is uncertain. The motility and absorption in the colon appear normal in the aged, in the absence of definite colon disease, but symptoms from diverticulosis of the colon seem definitely to increase with age at and after the fourth decade. This may indicate atrophy or decreased tone of the colon musculature or decrease in the elasticity of the connective tissue layers of the colon. According to Dr. Ivy, "Death in the aged is apparently only rarely due to the wearing out of the organs of the digestive system. In the absence of gastrointestinal cancer or local toxic or infectious processes, the digestive system seems capable of functioning beyond the ordinary life span."

THE CARDIOVASCULAR SYSTEM

Failure of health to the point of death, primarily due to failure of the cardiovascular system, is known to increase with age, at least beyond the fourth or fifth decade of life. In persons past sixty years the capacity of the heart to increase in rate and strength of beats during intense physical work is usually diminished. At the same time both cardiac and blood-vascular efficiency may persist in man to a degree rendering life possible, if not efficient, beyond the age of one hundred years. But in comparison, either the hereditary time clock is shorter or the accidents of living are more serious in the case of the cardiovascular system than in the case of the digestive system. Also, at present, it is somewhat more difficult to separate pure age factors from accidental disease in the cardiovascular system.

Instead of atrophy with age, there is an actual increase both in size and weight of the heart with advancing years, even when the increased fat in the heart of old people is deducted. But this does not mean increased efficiency. In fact, it may be secondary to cardiac incompetency. There is decrease in the elasticity of the heart valves, and increased calcium or cholesterol deposits in the valves. There is increased thickness of the endocardium, there is an increase in brown pigment in the muscle cells, and a diminution of the cross striation of the muscle cells, especially close to the nuclei.

On the functional side, cardiac irregularities, such as premature contractions and auricular fibrillations, show a tendency to increase with advancing years (see Chapters 24 and 25). Cardiac irregularities from hyperthyroidism are more frequent after the age of forty five. While this may be an indication of decreased cardiac reserve, it may also mean an increased incidence of hyperthyroidism beyond that age. There is a decrease in oxygen consumption in the heart with advancing years, the heart in this respect showing a parallel with the gradual lowering of the basal metabolic rate of the body as a whole. The electrocardiogram shows lessened voltage and some slowing of conduction in old people. This probably indicates some impairment of the conduction system. On the other hand, there appears to be an increased sensitivity of the carotid sinus cardio inhibitory reflex with the advancing years.

Where spasm or atheromatosis of the coronary arteries intervenes, we obviously have the same impairment of the heart as occurs in all other tissues where the efficiency of the circulation is impaired, by chronic functional spasms of the blood vessels, or by mechanical narrowing of the lumen, and by decreased elasticity associated with calcification of the blood vessel walls (see Chapter 27).

The old saying that the person is as old as his arteries can still be accepted as partly true. There is decreased elasticity of the aorta, decreased elasticity and increased calcification in all arteries in all people with advancing years.

But the rate of decrease in elasticity and the factors do not appear to be the same in all people. The rate of decrease in elasticity is not the same in all people.

we are faced with the problem. How much of these arterial impairments are due to the aging *per se*, and how much to the accidents of living? If it should turn out, as the work of L. R. Dragstedt indicates, that one main factor in the genesis of arterial sclerosis is faulty fat metabolism induced by the deficiency in a hormone or hormones from the pancreas necessary for normal fat metabolism, we are well on the road to a partial understanding and possibly prevention of this well nigh universal corrosion of a vital link in the life chain of the individual. Snapper suggests that the practical absence of meats and milk in the dietary of the people in North China may be a factor in the low incidence of arterial sclerosis in that population. In view of the work of Dragstedt, the over-all low caloric diet in that part of China may be a more significant element.

Owing to the gradually decreasing arterial elasticity with increasing age, there is a gradual increase in the speed of propagation of the arterial pulse.

of so-called "essential" hypertension and in the absence of an excess rate of the heart beat. Some investigators have estimated this rise in systolic blood pressure beyond forty as around 1 mm. of mercury per year (see p. 464). This is, of course, subject to a great deal of individual variation and to many exceptions, since the systolic blood pressure depends not solely on the condition of the arteries, but also on the efficiency of the heart and of the cardiac regulatory reflexes. Learning from the experience of the arteriosclerosis in the human

glomeruli with advancing age has been described (see p. 74).

THE KIDNEYS

The kidneys, like the digestive system and the cardiovascular system, serve the entire physiologic economy of the individual. Hence serious age impairment of either necessarily impairs the function of all. The kidneys show progressive reduction in weight after the fourth decade. The rat's kidneys show progressive involution of the glomeruli after the fourth month of life. In some fish the glomeruli disappears entirely in later life, and the renal organ becomes an aglomerular kidney (see p. 79). The one-year old rat exhibits

Nider has shown that the kidneys of old animals display increased susceptibility to injury by poisons (ether, chloroform, uranium), and decreased power of regeneration after such injuries.⁵ But to what extent this impairment of growth and repair is primarily a matter of the hereditary renal time clock or is aggravated by the accidents of living is still an open question. As to the kidneys and kidney function with advancing years, there is some evidence of actual decrease in quantity of renal tissue as well as in some renal functions, even in the absence of the recognized types of renal pathology. (See Chapters 4 and 37.)

In the aging individual man, renal involution or atrophy (tubular and glomerular) appears to be secondary to impairment of renal blood flow due to structural involution of the renal blood vessels. But despite such renal histopathology of the aging man, the kidney usually has sufficient reserve, in the absence of specific renal disease, to meet the requirements of the individual at the age of one hundred years and beyond. It is now well established by Goldblatt and others⁶ that marked and chronic decrease in renal blood flow is *one* factor which initiates functional hypertension of renal origin. Is this renal ischemia initiated or merely aggravated by the accidents of living? That is, is it an unavoidable turn of the renal time clock of the individual? The fact

type of renal failure. But it is clear that the serious corrosion of the renal life link, by inducing chronic hypertension and further impairment of the circulation, will seriously accelerate the depletion of reserves in all other organs and not only shorten the life span but seriously weaken the life of the aged, years before the death of the individual (see Chapter 30).

THE NEUROSKLETAL SYSTEM

The gradual slowing and weakening of reflexes and general body activity in the aging mammal are so obvious as to be well known to both physicians and laymen. Decreased functional capacity, both in the nervous tissues and in the skeletal muscular tissue, seems to be at the base of this gradual decline. Actual atrophy of the Purkinje cells of the cerebellum has been described in the aged and, since this part of the nervous system is seriously concerned with skeletal muscle tone and coordination of skeletal muscle contractions, it may be a factor in the growing muscular weakness of old people, irrespective of the cause or causes of this atrophy in the cerebellum.

As regards the cerebrum of aged people, general atrophy has been described, especially in the frontal and occipital lobes, and actual disappearance of cells in some of the layers of the cerebral cortex, as well as pigmentation and fat infiltration of the nerve cells and hyperplasia of the neuroglia cells. Similar degenerative changes with age occur in the spinal cord, that is atrophic pigmentation, actual loss of cells, and degeneration of the axones of many ventral horn cells. In the case of the brain, thickening of the meninges occurs with advancing age, but it is difficult to see how this in any way should interfere with nervous action or nervous function.

Recent investigations appear to demonstrate a very gradual but significant decrease in the myelinated fibers of the dorsal nerve roots with advancing age. This must be secondary to an atrophy and death of spinal ganglion cells and is probably the basis of the reduction in cutaneous and protopathic sensibility of aged people (see p. 288). The sense of pain seems to be the least affected by aging. In view of such evidence of atrophic and degenerative changes in the central and peripheral nervous system, irrespective of the primary cause or causes of these changes, it is not surprising that neuromuscular weakness, slowing of the reaction time, decreased capacity to learn, etc., are part and parcel of the physiology of aging. The speed of learning seems indeed to decrease gradually in man from the fourth decade on (see Chapter 5). But this handicap of the aged is on the whole more than made up for in their greater speed of correlation and evaluation of the new experience.

There is very little evidence of aging changes in *smooth muscle*. Smooth muscle seems on the whole to retain its normal histologic character into advanced old age. The diminished tone in smooth muscle, as may be seen in the blood vessels, in the gut, and in the smooth muscles of the skin and other structures in old people, may be secondary to the impairment in the nervous systems, as indicated above. But not all the impairment of body motility with age can be ascribed to the degenerative changes in the nervous system itself, because the striated skeletal muscle system shows fatty infiltration and brown atrophy with advancing age. The strength of the biceps at the sixth decade of life is only about 50 per cent of that at the age of twenty-five to thirty. The trunk muscles decline in power somewhat more slowly. There is some increase in connective tissue and elastic fibers in the skeletal muscle of old people and there is clear evidence of desiccation, that is, decrease in intracellular fluid. But in this respect, the skeletal muscle of the aged falls in line with all the other tissues of the body.

While the aging individual usually maintains an average normal body temperature, he adjusts less rapidly and completely to temperature changes in his environment, probably mainly because of the low metabolic rate and

atrophy of the skeletal muscles, as well as impairment of the circulation and sweat secretion

VISION AND HEARING

Because of the accessibility of the sense organs themselves and the availability of quantitative tests of functions, we have more accurate information regarding some of the aging changes in the physiology of the eye and of the ear than is the case with most of the other systems in the human body

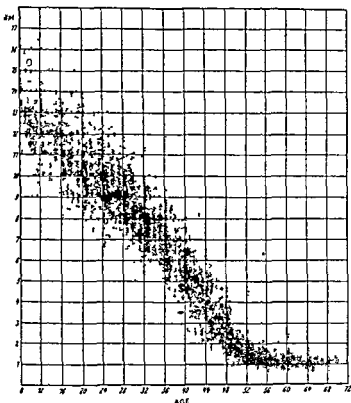


Fig 14 Range of accommodation in relation to age in 4000 cases (Duane)

Vision In the case of vision there is a gradual decrease in visual acuity (central vision) and a gradual narrowing of the visual field, as well as a slowing of the dark adaptation (peripheral vision) and a gradually higher threshold for light stimulation for man past the fourth decade. The narrowing of the visual field is probably due to the actual degeneration of the nerve cells, starting in the periphery of the retina. We are not yet in position to say whether these visual impairments occur independent of or are secondary to impaired retinal circulation. But it seems significant that anoxia decreases peripheral vision in young people.

It is well known that the incidence of cataract increases with age, irrespective of whether or not the tendency to cataract formation is hereditary. Arteri-

osclerosis would undoubtedly accelerate any such hereditary weakness, and so would certain faulty diets and certain endocrine and other metabolic disorders

The gradual decrease of the elasticity of the lens is another well known and accurately measured phenomenon of aging man, with this exception that diminution in lens elasticity actually starts in childhood and practically all lens elasticity is lost before sixty years of age. The lens continues to grow at the periphery (vertex) and thus approaches closer and closer to the cornea with advancing years. At the same time the material at the center of the lens becomes more dense. These factors, plus the swelling of the lens from increased water, are responsible for the well known phenomenon of so-called "second sight" of people sixty years of age and older. This lens change tends to counteract the impairment of accommodation, due to the loss of lens elasticity (See Chapter 19)

Other age changes that may contribute to the gradual impairment of vision with age are diminished translucency (arcus senilis) of the cornea and of the vitreous humor. It need not be pointed out that the retina, being actually a lobe of the brain, is necessarily as seriously impaired by local vascular pathology as is any other part of the brain. And because of the accessibility of the retinal vessels to direct inspection, we have probably earlier factual information regarding such pathology in the retina than we have in most of the other deep organs of the body (see p. 294)

Hearing. From the age of about twenty on there is a gradual loss of acuity to all tones, but the loss of sensitivity is greater to the high tones. This deterioration of hearing is somewhat greater in the male, but the degree of retrogression is not predictable on chronologic age, as some people at eighty have no greater auditory impairment than other apparently normal people at fifty. This impairment of auditory acuity is present even when tested by bone conduction. The cause for this decline in auditory acuity appears to be a gradual but distinct atrophy of the nerve cells in the basal coil of the cochlea. But local anemia, due to incipient arteriolar sclerosis, may also be a factor, since in experimental anoxia, the perception of the high tones goes out first.

We have at present no extensive information regarding impairment of vestibular reflexes with advancing years (See Chapter 20). One would expect such impairment on the basis of the evidence of age atrophy in the central nervous system.

THE ENDOCRINE SYSTEM

Gonads. Owing to the relative ease of observation as well as experimentation and measurements of such phenomena as menstruation, size and function of secondary sex characters, libido, sex behavior, and the like, we have the clearest information, at least regarding the factor of aging, in the case of the ovaries and the testes. In the case of the gonads, we have the additional special phenomena that, irrespective of their probable role as hormone producers

chain of the individual, their absence does not shorten the life span. Prolongation of youth, or of life itself, by gonad hormone therapy is a mirage, a product of wishful thinking.

The rate of decline of the human ovaries at the normal menopause may

be as speedy as a few months or may be prolonged several years, this varying with the individual woman. When ovarian atrophy appears much earlier than the thirty fifth to forty fifth year, one can usually find specific diseases contributing to such decline, although we cannot with certainty eliminate specific hereditary weakness in the constitution of the individual, except where destructive tumors or serious impairment of thyroid or pituitary functions are clearly involved. The life span of the testes is longer and the decline is more gradual. The hormone production mechanism in the testes, the factor involved in libido and copulation, appears to fail earlier than the spermatogonia and the sperm producing factors. At least in individual instances in the human male, sperm production (not necessarily normal sperm) has been described up to, if not beyond, the ninth decade of life. In the aging human testes the basement membrane of the tubules is reported to grow thicker, and the spermatogonia change or retrograde into the small round cells seen in the immature testes.

In the case of the interstitial cells, increased pigmentation has been described in old testes. But there is at present no evidence of decrease in the number of interstitial cells. It is difficult to interpret the usual decrease in libido and sexual potency in the aged human male as related to the gradual atrophy of the testes, in view of the fact that in some males between twenty and thirty five libido and sexual potency appear to persist even after total castration. In other words, the mental and vasomotor conditioning associated with the above physiologic processes apparently can persist in the total absence of the primary conditioning factor, the male hormone.

Increased pigmentation and gradual atrophy of the acini of the prostate gland have been described as occurring in old people and old males of other species, but there appears to be no decrease in gross size of the prostate, despite the fact that castration in the young male leads to atrophy of the prostate and its adnexa. We are here facing the well known dilemma in this physiologic system, namely, the dependence of the prostate on the male hormone during early adult years, the relative independence of the prostate to the male hormone during the later decades of life, and the frequent appearance of actual prostate hypertrophy parallel with the apparent decline of testicular hormone production in old men (see p. 251).

Thyroid. We have evidence of some shrinkage in size and some evidence of cellular atrophy in the thyroid in old people and old animals. Some people have inferred that this is a factor in the decreased oxidation of tissues with advancing years, but since the thyroids of youth and adult life have such large factors of safety or reserves, we must have more direct evidence than this for

Pancreas. In the case of the pancreas and insulin, the evidence of increased histopathology in the pancreas with advancing years, the fact of increased incidence of actual diabetes, at least up to the age of fifty or sixty, as well as the gradual decrease in sugar tolerance with advancing age, as evidenced by the glucose tolerance tests, all point to some aging in pancreatic physiology, in the insulin producing mechanism or the rate of response of these mechanisms parallel with the advancing years. The perennial puzzle in this field is the evidence, in such animals as rabbits, guinea pigs, and dogs of

the great regenerative power of the pancreas, regeneration both of island tissue and the pancreatic juice-producing cells, and the apparent absence of such regenerative power in the pancreas of the human species, at least in those people whose pancreatic reserves have, through heredity or the accidents of living been reduced to the point of actual diabetes. But whether we are here dealing primarily with aging or with accidents of living, it is nevertheless a fact that, in some individuals at least the hormone link of the pancreas may stand the wear and tear of living up to and beyond the age of one hundred years in man, provided the pancreas reserves have survived or met the needs up to the age of fifty or sixty (See Chapter 14)

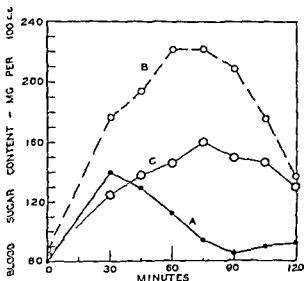


Fig 15 Age changes in glucose tolerance curves A Blood sugar levels after ingestion of 50 gm glucose by forty year old male B Blood sugar levels after ingestion of 50 gm glucose by eighty year old normal healthy male Curve rises slowly to higher levels than in young adult, indicating impairment of removal or storage mechanism C, Blood sugar levels after ingestion of 50 gm glucose by eighty year old normal healthy male Curve shows slow

Press)²

The work of L. R. Dragstedt and his associates shows that the second pancreas hormone or hormones (lipocaic) as a fundamental factor in normal fat metabolism may be a more important element in the aging processes than is insulin, for fatty degeneration or infiltration of cells and tissues (such as the blood vessel sclerosis) seems to be an almost universal phenomenon in the aging and the very old animal. We have no information on the requirements of lipocaic in relation to age, or the ability of the aging pancreas to supply this necessary factor.

Pituitary. Structural changes have been described in the pituitary glands of old animals as well as old people, but in the absence of specific pituitary disease, this endocrine gland appears to be remarkably stable at least up to

the age of eighty or one hundred years. There is no evidence, at present, for ascribing the early failure of the ovaries, the later failure of the testes, and the alleged atrophy and hyposecretion of the thyroid in advancing years to decreased production of the gonadotropic and the thyrotropic hormones respectively (see p 249)

Adrenal. Armchair biologists have little difficulty in pointing out the almost perfect parallel between the symptomatology of aging and that of impaired adrenal cortical function, such as general asthenia, impaired digestion, increased pigmentation of the skin, and low basal metabolic rate. Thus one might make out a plausible case for gradual adrenal cortical failure as a causative factor in aging. But the usual arterial hypertension with advancing age does not fit this picture, and significant adrenal cortical failure is always associated with disturbance in the balance of the inorganic salts of the blood plasma, a phenomenon which is absent in old people. There is at present no evidence that deficiency or excess in adrenal hormone production is a significant factor in aging.

Parathyroids. The parathyroid hormone is important in the physiology of bone and in the regulation of the calcium and phosphorus of the blood. The latter is usually practically normal in old and very old people. The bones of the aged are not normal, but obviously we cannot ascribe the bone impairment of the aged to either hypo- or hyperfunction of the parathyroid glands (see p 254).

Thymus. The thymus was once classed with the endocrine glands and it may indeed have some endocrine function in the fetal and preadolescent life of man, as well as in animals below man (possibly the birds). But after adolescence or body maturity, the thymus becomes essentially a lymph gland. The findings of Rowntree and associates that injection of thymus extract induces accelerated growth and precocious sexual maturity in rats have not been substantiated, and the still earlier work indicating that the thymus may be necessary for the life of bone, if not for the life of the individual animal, was long since wiped off the board by the conclusive work of Park and McClure. Indeed, the so called "persistent" thymus was once thought to be the cause of sudden death.

BONES, TEETH, AND CONNECTIVE TISSUE

A gradual solution and absorption of bone in old mice has been described. In man, the bones become more brittle with advancing years. This may be due to a change in the chemical combination of the bone calcium, a change towards a more amorphous state. The demineralization of bone that proceeds with advancing age does not go on with equal rate in all the bones of the body. Albright attributes the osteoporosis of senility to the loss of gonadal hormones and diminution of the adrenal cortical "N" hormone. He is convinced the osteoporosis responds to estrogen or testosterone therapy.⁶ Todd is inclined to charge the age changes in bones and connective tissue to arteriosclerosis, that is, to impaired circulation and nutrition. There is a gradual reduction in the red bone marrow in the vertebrae, and decrease of the lecithin in the bone marrow with advancing years. Parallel with these struc-

proceed along parallel lines. In true tendons and ligaments, age changes, apart from those due to specific disease and malnutrition, have not been made out.

In the case of *teeth*, decreased permeability of the enamel, thickening of the secondary dentine, and increased calcium content with age have been reported. In some herbivora, an actual mechanical wearing out of the teeth from mastication occurs. Such mechanical wearing out is surely a minor factor in modern man. The decay and failure of teeth with age in modern man is probably more a matter of faulty nutrition and other accidents of living than the rapid ticking of the hereditary time clock.

LYMPHATIC SYSTEM

The changes in the number and kind of lymphocytes and leukocytes with age do not seem significant. These cells are individually shortlived and the tissues producing them seem to be able to do this job adequately, at least up to the age of ninety to one hundred years. Nevertheless, there is some evidence of atrophy and fatty infiltration of the lymph nodes in old animals and old people. The lymphoid tissue in the vermiform appendix decreases with age. There is some decrease in size of the tonsils and adenoids in people fifty to eighty years old, and a similar but slight decrease in the lymphoid tissue of the spleen. On the whole, both the aging factor *per se* and the accidents of living appear to deal lightly with the lymphatic system.

THE BLOOD

The blood of the newborn and the very old is almost as accessible to quantitative study as are the more superficial tissues of the human body, and such studies have grown apace in the last fifty years, driven not so much by the curiosity concerning aging as the curiosity and necessity for information in relation to specific diseases. Increased fragility of the erythrocytes with age has been reported. But in the absence of specific disease, this is not sufficient to cause anemia in old age. (See Chapter 13.) Indeed in the entire blood system, in both cell count and cell character, in plasma volume and plasma composition, there is a greater constancy and uniformity in this tissue throughout the entire span of life than in the other organs of the body. It is, however, probable that all the subtle age changes in the blood of man and other animals have not yet been discovered, because of lack of adequate methods. It seems to be a fact that the serum of old chickens retards or inhibits growth and induces *fatty degeneration in connective tissue cells in tissue cultures*, in comparison with plasma from chick embryos. Nevertheless, in the absence of serious impairments in diets, and in the renal, respiratory, circulatory, and endocrine mechanisms, the fundamental homeostatic mechanisms in which the blood plays such an important part (body temperature, acid-base balance, blood

THE SKIN

The common changes with age in the hair, skin, and nails are familiar even to laymen. In the case of the hair on the human scalp, it is also known that acute infections, disorders of metabolism, and endocrine disorders intensify these involutional changes. The skin changes include increased pigmentation of the exposed parts, decrease of water, decrease of fat and decrease of elasticity in the skin, as well as decrease in its growth and regenerative capacity. A skin wound of 40 sq. cm. in a person twenty years old heals on the whole in forty days. The same size skin wound in a person forty years old requires for healing about eighty days. At the age of sixty the same skin wound requires five times longer to heal than in a child of ten. In this regard the skin follows the descending curve of regenerative capacity with age seen in practically all the other tissues of the body (see p. 79). The decrease in skin elasticity appears to run parallel with the actual degeneration of the elastic connective tissue fibers. The decrease in the subcutaneous fat may play a minor role. The dryness of the aged skin is probably secondary to decreased secretion of the sebaceous glands, although these glands show, on the whole, hyperplasia with the advancing years (see Chapter 44).

On account of its cosmetic significance for modern man, the failure of color and the gradual falling out of the hair of the scalp have received a great deal of unscientific attention. There are indications of an hereditary factor. Diet, disease, and vascularity unquestionably play a role, but in the absence or with the minimum of these factors at play, there seems no doubt that the hair on the scalp at least would sooner or later run its course of graying and death, considerably before the zero hour of the time clock for the individual person.

The complexity and the individuality of some of these mechanisms, hereditary and accidental, are indicated by the fact that parallel with the graying and falling out of the hair on the scalp, some of the body hairs, such as the eyebrows, the hair in the nose, and on the external ear, may show an increased growth if not an increased pigmentation with the lengthening shadows of the accumulated years.

DIET AND LIFE SPAN

Since the scientific information on the kinds and the quantity of foods required for growth and health is very recent, considering the history of the human race, and this information is still both fragmentary and inadequately applied, it is evident that in the past man's diet, both as to kind and quantity, was determined by appetite, hunger, and the availability of the foods. We may, therefore, assume that under consumption and excess consumption of foods, starvation and gluttony, emaciation and obesity, are phenomena as ancient as man himself.

Life insurance statistics show clearly that underweight as well as obesity tends to shorten the life span of man. McCay¹ finds that with a diet adequate in quality, but so inadequate in amount fed that the growth of the animal is practically at a standstill, the life span of rats is significantly prolonged. In this experiment the control group of rats was fed the same kind of food *ad libitum*, so that this group may have had its life span shortened by excessive eating, that is, by obesity. It is well established that many, if not all, persons showing marked and persistent underweight or emaciation have functional disorders

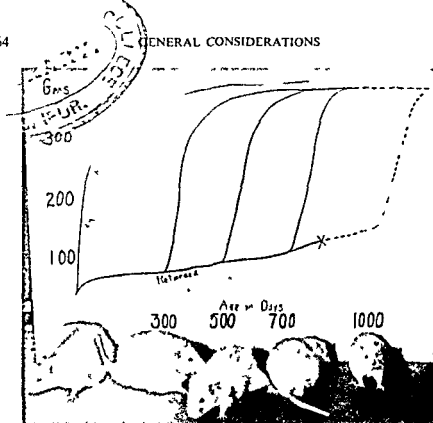
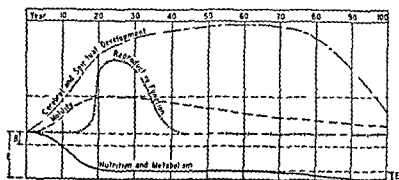


Fig 16 These rats are almost the same age about 800 days The background of growth curves shows the age at which each matured The young one on the extreme right is still awaiting its opportunity to complete its growth Although the rat on the left represented a group twice as large as any of the others at the start of the experiment it and all other members of this normal group were dead at the end of 965 days while some members of each of the retarded groups were still alive (McCay in Cowdry Problems of Ageing Williams and Wilkins Co)¹



interfering with the digestion, absorption, or metabolism of foodstuffs, excess metabolic rate, or chronic disturbance of appetite and hunger (anorexia nervosa). That such persons die prematurely presents no particular problem, since we have here clearly an excess corrosion of essential links in the life chain. But why does the overweight person, the obese person, who seems otherwise healthy, die prematurely? This is a fundamental problem in geriatric medicine, for in the absence of specific disease, overweight or obesity tends to increase in men and women past forty years of life. Is it a disorder, a disease? Or is the shortening of life span by obesity, as implied in the popular saying, "fat is the enemy of life," a natural consequence of the process?

There may be some interference with respiration and circulation from the increased pressure of the abdominal organs against the diaphragm. Admitted that there may be hereditary factors in some species, such as the whale and the hog, favoring deposition, or excess deposition, of body fat, it is nevertheless true that all animals, otherwise normal, can be fattened by eating in excess of the expenditure of energy. In man obesity is the result of the same funda-



LIFE CURVES OF VITAL FUNCTIONS

Fig. 18. Approximate curves of some physiologic processes in man and their changes with age. (After Stritz, from Warthin, *Old Age*, Paul B. Hoeber, Inc.)¹⁰

mental cause, that is, eating in excess of the energy expended. The work of Newburgh goes to show that intake of food below the energy requirements

fourth day from the day of weaning until the day of death lived significantly longer than the rats eating the same diet *ad libitum* every day. This amount of fasting did not significantly retard growth and body size as measured by tibia length. But the rats on this type of fasting did not become obese. On days when these rats had no food they were usually more active and restless than their litter mates on the same food *ad lib*. It seems probable that the rats given all the good food they chose to eat every day shortened their life span by overeating rather than that the fasting and greater physical activity of their litter mates actually lengthened their genetic life span. For when considerable indigestible bulk was added to the same diet and the rats were allowed to eat this every day the life span of these rats was also lengthened.

During the last twenty-five years considerable, though superficial, attention has been given to the possible relation of thyroid, gonad, hypothalamic, and pituitary disorders to human and animal obesity. It now seems established, or at least highly probable, that there is no such thing as a primary endocrine obesity, apart from endocrine influences on appetite, hunger, and physical activity. The marked obesity that may follow destructive injuries at the base of the hypothalamus seems to be largely due to increased food consumption because of increased appetite. Endocrine therapy of obesity has been disappointing, except in the case of thyroid extract, and the latter has to be administered to the point of inducing the initial symptoms of the disease, hyperthyroidism, before weight reduction is secured, in the absence of reduced food intake. On the basis of present information it seems likely that the development of obesity in some people past forty years of age is primarily due to the persistence of these people in excessive eating and the satisfaction of specific appetites for foods, when the rate of tissue oxidation and general motor activity are both on the decline.

It is not difficult to understand why ingestion of food to the point of obesity is injurious to people with reduced factors of safety in the matter of insulin, and of sugar and fat metabolism. Such dietary excesses damage by overwork an already impaired mechanism. But in the absence of diabetes, actual or incipient, why does obesity, maintained for years, initiate or aggravate cardiovascular, renal, and other disorders that shorten the life span? While the answer to these questions is being sought by experiments and accurate observation on mice and men, the prevention of obesity in all people past thirty appears to be a prophylactic imperative, a "must," in preventive geriatrics.¹¹ And as a general proposition, it is far safer for the patient to do this by reducing the diet than by taking toxic drugs. (See Chapter 6.)

SUMMARY

Except for immunities to specific infection and the pathologic hyperplasias and tumors, all the changes in the machinery of the body which parallel, and, in fact, constitute the aging process, point to a *gradual depletion of the hereditary tissue reserves or "factors of safety," so that the aging individual becomes gradually less able to meet the usual accidents and the unusual stresses of living.* As long as these can be avoided, life goes on until the corroded life links break, even in the absence of specific storm and stress. But this way out appears to be a very exceptional event in our own species.

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CHAPTER 4

ANATOMIC CHANGES OF NORMAL AGING

JEAN OLIVER

THOUGH no pathologist, however dynamic his concepts, can well avoid the ultimate admission of the essential structural determination of functional disturbance, it is certain that the visible morphologic alterations which mark the passage of years cannot be regarded as the source of the senescent process. Even the more or less changes, cell loss atrophy, for example, long described very bal-

ance. In fact the biochemists have demonstrated such shifts long before any structural change is visible in the disturbed tissues¹ of alterations more grave than those of senescence. It is helpful therefore to the writer, a morphological pathologist, that his exposition has been preceded by a description of the functional disturbances of aging and it seems to him proper that his first reference is to the work of the biochemist.

The present chapter proposes to describe only the more important of the structural changes that come with apparently normal aging and to leave to individual authors a description of the special pathology of the systems with which they are concerned. Before detailing these changes, however, more general considerations will be helpful.

NORMAL VS. PATHOLOGIC CHANGES

The first of these considerations is that none of the structural changes observed in aging is peculiar to that state. They are repetitions of alterations that are familiar throughout the life of the organism. They have operated with evolutionary effect both in its phylogenetic and ontogenetic past. In the kidneys of certain fish are found glomerular atrophies with tubular persistence² not unlike those seen in the senile human kidney,³ and in the latter may be found evidences of tubular disruption into cysts identical, not only in its effect but in its vascular origin, with what has occurred in the mesonephros of the same individual before he was born.⁴ The same processes, or at least the same structural evidence of the involutions by which the individual has developed, are therefore found in what Warthin⁵ has so aptly called the "major involution."

These natural and therefore "normal" and proper involutions do not, however, comprise the whole picture of senile structural change. There are the myriad "pathologic" alterations which add their quota of disintegrating effect and in the sorting out of these from what is appropriate to this chapter difficulties of definition and concept arise.

To begin with a relatively simple aspect of this problem of the "normal" and the "pathologic," no one has seen the lungs of an elderly individual without some evidence, if only microscopic, of a bronchopneumonia. Yet it would

be obviously illogical to describe exudation of fibrin and leukocytes as part of the senescent process. Many other examples of chronic infection that regularly form a part of old age might also be cited. Here the introduction of the extrinsic factor excludes the intrinsic, but with another series of structural changes so constantly found in the aged as to be universal the answer is less simple—for example, the results of the wear and tear of life.

Both the door hinge and the shoulder joint wear rough and creak, not because they are old, but because their age has given rise to much use. A truly senescent change has not occurred, though the passage of time was essential, if the individual is leading an average existence, for the accumulation of much effect.

The whole problem of wear and tear, on its face so simple and mechanical, becomes indeed progressively more complex as one considers it. In the arterial system for example, and in the same vascular segment, the aorta, it is seen not only as a primary factor in vascular alteration, as evidenced by the predilection of atheromatous infiltration at *points of strain* where the vessel divides, but as a secondary or auxiliary factor that leads to the development of such structural change as ectasis, which is the direct result of another factor, namely senescent degeneration of elastic tissue. And finally how much wear and tear is to be allotted as the proper or "normal" part of an individual's existence and at what point is it to be considered excessive or pathologic?

Any mention of the vascular system introduces another series of similar difficulties into the problem of normal senescence. The first gasp of extra-uterine life accelerates the involution of certain arteries, the ductus arteriosus and the hypogastrics, so that changes are found in the arteries of the newborn not dissimilar to those seen in the aged.⁶ The latter must be regarded therefore as a part of a normal involution, but mixed with these components in the first year of life are other alterations, such as fatty deposition⁷ that result, when time is sufficient, in scarring parenchymal atrophies or more disastrous thromboses and ruptures.

It would save confusion if we admitted frankly that normal and pathologic are not sharply demarcated, that they are often indistinguishable. We must after all accept the aged individual as a whole with all his infirmities and not hold to some theoretical ideal that we shall never meet—and this of course is what we all do, except when writing learned treatises. The definition of normal structural changes made in this chapter will therefore be in the statistical sense, if most old individuals show a certain change, that change will be accepted as an entirely proper reaction of the individual to the environment that was his and therefore normal to him, and no attempt will be made to set apart some of these reactions as pathologic. The more important of these frequent alterations may now be described.

PARENCHYMAL ATROPHY AND INTERSTITIAL INCREASE

The fact concerning these alterations is simply stated. In most of the

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In most instances, however, it seems impossible to consider separately this twin pair of structural alterations. Ever since Weigert and Virchow argued the primacy of one over the other, no entire agreement has been reached as to their relative importance. In general perhaps the theory of Weigert, that the decrease in size of the parenchymal elements precedes the increase, which is at times admittedly only relative and therefore apparent, of the interstitial tissue, seems a satisfactory description of the time relations of the two changes. As will appear in further discussion, however, the interstitial change is only rarely a purely passive alteration. Loeb⁸ describes the senescent increase in the stroma of the thyroid as a primary reaction with secondary effects of pressure and interference with the nourishment of the parenchymal cells that leads to their atrophy. A further discussion of this problem may be left to a later general consideration of the effect of hormones on the structural changes of senescence.

Causes. Granting the importance of the atrophy of the parenchymal cells, difficulties arise in the consideration of its origin. Can this be found in an inherent and primary failure of the cell to maintain itself, to which Warthin attributes most of the phenomena of the major involution of senescence and which he dates as first visibly evident with the penetration of the spermatozoon into the ovum? Such an "abiotrophic" atrophy, in the writer's opinion, is not redeemed by vague allusion to entrophy and to the second law of thermodynamics from its fog of vitalistic mysticism. Moreover there is no apparent loss of growth energy when cells even so highly differentiated as those of vertebrates are isolated from the aging environment of the individual, for they show no loss of ability to grow and to multiply but maintain themselves indefinitely in tissue culture.⁹

One might look therefore to the environment of the atrophic cell for the cause of its failure. Accumulation, both intra- and extracellular, of products that act upon the metabolism of a cell is conceivable, and suggestive experiments have indicated the presence of such substances in the plasma of old individuals.¹⁰ Such accumulation is also a possible source of the interstitial alteration which at times seems to precede the cellular atrophy. One thinks of the infiltrations with hyaline substances, the swelling and hyalinization of collagen fibrils and basement membranes and the deposition of calcareous material, all alterations that can be conceived as a part of change in the *milieu interieur*. It is along this line of investigation that the biochemical attack offers such promise to the understanding of structural changes of aging.

In all its aspects the *metabolic environment* is dependent on the circulation of the blood. Interference with vascular supply, both of inflow and outflow, and also with the ebb and flow of lymph is a definite and relatively clearly comprehensible example of an environmental cause of cell atrophy. In the human senile kidney for example, it is indeed rare that the factors of decreased nutritional effect due to involutional arterial change are not sufficient to account for all the atrophy of nephrons that constitutes the senile scar.³ In the same individual, however, the brown atrophy of the liver, an organ of double blood supply, seems difficult to explain on the basis of the moderate sclerotic changes that are seen in the small arterioles. That this antithesis is not peculiar to differences in organ reaction, moreover, is shown by the occurrence of renal atrophies in senile dogs where the arterial system is relatively unaffected.

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PARENCHYMAL ATROPHY AND INTERSTITIAL INCREASE

The fact concerning these alterations is simply stated. In most of the organs of old individuals one sees an association of decrease in the size of the functioning parenchymal cells, with an increase both in amount and in density of the interstitial substance. As in all biologic descriptions, an exception must be mentioned at once, where dissociation of the two closely related lesions is regularly observed. Such is the situation in the liver, for here parenchymal atrophy is not regularly associated with any considerable interstitial increase.

contour that are typical of the senile organ. That the atrophy of the nephron is dependent upon the obliteration of the glomerulus in an analogous manner to that of the axon on its nerve cell is a fallacy that has succumbed to a method of direct examination of the problem. There is no correlation between the structural state of the glomerulus and its renal tubule.¹²

Combination of Atrophies Another reason for the extreme effects that atrophy produces in aging tissues is the frequency with which several kinds or

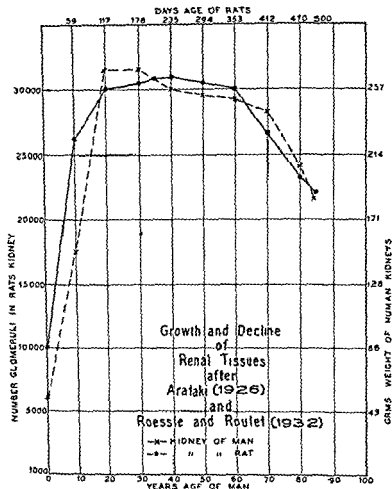


Fig 19 Growth and decline of renal tissues (After Arataki, Roessle and Roulet)

manners of decrease in size may be combined in a single organ. It is customary to label these different alterations as various sorts of "atrophy," a classification that perhaps presupposes more exact knowledge of their causes than we actually possess. The aged heart, whether large or small, constantly shows such combination of atrophies. There is first the diffuse, so-called "simple" atrophy with pigmentation that is often considered to be evidence of senescence *per se*. There is little, or at least only relative, increase in the connective tissue stroma between the shrunken muscle cells. In other regions, part

in the ventricular walls, are found areas where the histologic picture seems the converse there is a definite increase and thickening of the fibrous and elastic stroma¹³ which infiltrates and surrounds the atrophied muscle cells. The suspicion that the origin of this fibrosis may be sought in a nutritional vascular disturbance becomes a certainty, when, as is frequently the case, definite areas of complete atrophic disappearance of muscle cells, with heavy collagenous replacement give evidence of arteriosclerotic scarring. It is the summation of these atrophies abiotrophic and nutritional, that is responsible for the extreme change in the organ.

Various combinations of similar kinds of atrophy are found in other organs. In the kidney of man, the predominant atrophy is of vascular or nutritional (arteriosclerotic) origin, but this is not true of the dog's kidney, where the simple form of decrease in cell and unit size occurs. In the liver, as has been mentioned, there is little evidence of arteriosclerotic atrophy, the change observed being apparently one of the clearest examples of simple atrophy. In the central nervous system the neuropathologists are able in most instances to recognize both primary senescent atrophy and the effect of sclerotic vascular change.

A special case of senile tissue atrophy where neither cell nor organ units are primarily involved in the decrease of organ size is seen in the wastage of bone that is so prominent in the aged. Here the process, affecting an interstitial substance, results in a demineralization or halisteresis of the bony tissue. The secondary result of this molecular disintegration, which Todd¹⁴ attributes to senile nutritional depression, is a disintegration of cell and trabecular architecture that leads to diminished size of the bone involved. The typical change of contour in the mandible is exaggerated by the loss of teeth in old age but the bones of the foot show the uncomplicated change early, with those in hand, innominate, vertebrae, sternum and ribs following.

Before leaving the problem of senile atrophy a word must be said of the *purported irreversibility of the process*. It is true that atrophied tissues and cells in the young show a remarkable ability to regain their former size and function when interference with their growth is removed. In the aged these interferences can never be entirely removed, for apart from any theoretical inherent senescent weakness in the cell itself, the environment with its fibrosis and vascular sclerosis of senile tissue is a factor sufficient to prevent recovery. In fact, the part of any abiotrophic inherent factor in this particular instance seems, as always, a dubious cloak for our ignorance, a cover of that residuum persisting after explanation by the comprehensible factors of environmental malnutrition.

still impeded by their aged environment. When freed of this by the technic of tissue culture they seem able to grow indefinitely.⁹

ACCUMULATIONS AND INFILTRATIONS IN SENESCENT TISSUE CHANGES

The gradual accumulation of ill defined substances, the "schlacken" (Ribbert) of the cells' metabolic fires, has been suggested as a source of products which might impede energy transformations in the cell and thus act as a cause of regressive senescent change. The chain of cause and effect in this

reasoning might of course be equally well reversed, a retardation of metabolic exchange by senescence, allowing an accumulation of waste products. But in either case, once established, even if only as a vicious circle, it could conceivably operate to effect cellular regression, both functional and structural. Apart from these more or less hypothetical considerations, accumulations certainly do occur, at times even in sufficient degree to become visible within the cell. At least such is the implication of the words "Abnutzung" or "wear and tear" that are commonly applied to the pigment found in most atrophied cells. "Brown atrophy" is the descriptive term applied to the typical senescent heart, liver, or kidney. Even in the central nervous system, where no general discoloration of the organ occurs, microscopic examination shows the atrophied ganglion cells to be deeply pigmented and that this discoloration is only masked by the opaque tissue of glia and medullated fibers that form the bulk of the organ.

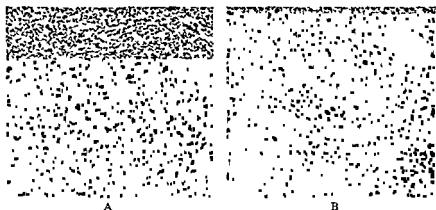


Fig. 20 Age changes in the spleen consist of atrophy of the lymphadenoid tissue with little increase in the connective tissue stroma, combined with a sclerosis of the terminal arteries. Contrast the large Malpighian bodies of the twenty-year-old spleen (A) with the small follicle of that of sixty years (B). Note the thickened central artery of the latter. Senescent arterial sclerosis occurs early in this organ and is therefore not necessarily indicative of an equally severe generalized arterial involvement.

The pigments observed in atrophied tissues are mostly lipochromes. Herein lies a source of confusion, for the exogenous carotenes taken in with the food and stored by certain tissue cells are chemically similar bodies. Lipochrome pigments are also found in certain cells early in life before any appreciable atrophy has occurred. The whole matter of wear and tear pigmentation therefore is filled with uncertainty. One sure fact, however, remains that no matter what the source or the significance of these pigments in the metabolism of the cells, an atrophied cell usually appears more heavily pigmented, even if that increase may be only relative to its smaller size.

Another accumulation, and one again involving the metabolism of lipoids, is seen in the infiltration of the walls of arteries. Atheromatous deposits, whether considered pathologic or normal, form in the first years of life⁷ as fatty spots in the intima and increase in size and extent to form the plaques that are universally present in old age. The fats concerned are the more complex lipoids, cholesterol and its esters, or phosphorus-containing

lipoids. The deposit may be in the form of droplets lying free in the interstitial tissue spaces of the intima of the vessel or in histiocytes of local or distant origin.

A certain variation in the occurrence of atheromatous change does occur. Some old arteries are relatively well preserved in this regard, and this fact is the basis for the separation by some of these changes from the category of the normal senescent change. The deposit of fat in the intima of the aorta is certainly dependent on many factors: the lipid content of the blood, both quantitative and qualitative, the condition of the tissue, this varying with the wear and tear to which it has been subjected and the state of preservation of its elastic element; and perhaps most important, the status of the individual's metabolic activity. All these factors operate in the everyday normal life of the individual and in varying combinations contribute to the production of varying effect. With the passage of time these effects may become significant by their accumulation.

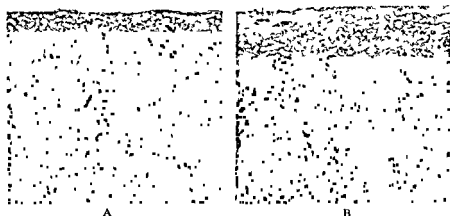


Fig. 21 Age changes in kidney are largely the result of senescent arterial sclerosis. Note how the even pattern of the twenty-five-year old kidney (A) is broken up by the large "arteriosclerotic scars" in the seventy year-old organ (B). Many well preserved glomeruli and tubules persist, however, so there is seldom clinical evidence of renal insufficiency.

Another infiltrative change of old age is the deposit of *calcium salts* in tissues. The calcification of the atheromatous artery can be considered no more pathologic than the deposit of infiltrating lipid, a fact emphasized by its occurrence in the wall of arteries where there are no atheromata. Faber¹⁷ derives later arteriosclerotic changes from such infiltrations with calcium that begin early and regularly and gradually accumulate with time to produce disturbances in the functioning of the media and thus a secondary development of sclerotic change throughout the vessel wall.

There can be no stigma of the pathologic found in the deposits of calcium in certain other senile tissues, notably the costal cartilages and those of the upper respiratory tract, larynx, trachea, and bronchi. Here the salts infiltrate the connective tissue of the cartilage and form dense, hyaline, amorphous formations as the corpora amylacea that are found in the central nervous system of the aging.

The mechanism of these depositions can be simply and, in part, adequately described as a *metastatic calcification* in Virchow's meaning of the term. The atrophy or wastage of the bones in the aging to which previous mention has been made frees the salt, which is again precipitated in areas of low metabolic exchange. These areas may be supposed to be particularly susceptible in the aged because the lowering of general nutritional activity has decreased the chemical exchanges, feeble in these tissues at best, that are presumed to keep in solution the calcareous materials.

Finally, accumulations of *interstitial mucin* are also found more frequently in the old than during maturity. Whether one can derive significance from the observation that mucin being the common interstitial substance of the embryo, its recurrence in the latter part of life may be regarded as evidence of an involutional dedifferentiation to simpler conditions, seems doubtful. Warthin⁵ speaks of a myxedematous stroma replacement that accompanies the senile atrophy of fatty tissue. In the media of large arteries, particularly



A



B

Fig. 22. A. Mucin in the interstitial tissue of the aorta. B. Normal tissue of the aorta.

sclerosis

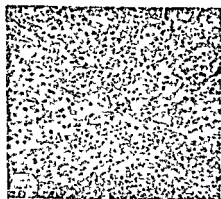
the aorta, collections of material presenting the histologic properties of mucus may accumulate to a degree sufficient to weaken the strength of the vessel wall.

ALTERATIONS IN THE COLLOIDAL BEHAVIOR OF TISSUE COMPONENTS AS THE SOURCE OF SENESCENT CHANGE

Many of the structural changes seen in senescence are the direct results of the fundamental characteristics of all colloidal systems, whether living or nonliving. The relation of the phases of which such systems are composed is by its nature that of an unstable equilibrium. It must shift and, therefore, time produces change, or aging. In particular, the relation of the hydrophilic colloidal particle to its surrounding aqueous phase is such that shifts of water from one phase to the other occur with resulting changes in the properties of the total system. "Functional" disturbance, without visible structural change, may result from aging, as shown by the decreased catalytic activity of an old

preparation of manganese dioxide over a freshly prepared solution, or obvious "structural" alterations are seen, as when an agar jell loses water and becomes granular. Living cells as colloidal systems can behave no differently and this fact is a most satisfactory basis from which a hypothetical consideration of the structural aspects of biologic aging can start.

Upon applying this concept to the structural changes seen in cellular atrophy one encounters a striking analogy. The protoplasm of the atrophic cell is decreased in volume and shows increased density so that on histologic examination it stains heavily. Exactly similar changes are seen in a mass of hydrophilic colloid gelatin as it loses water, shrinks, and becomes granular. An even more remarkable similarity is found in the comparison of the colloid rubber with the tissue element elastin, for with both substances the physical property of elasticity finds functional application. The reader is referred to Wells' remarkable development of this analogy in his comparison of arteries and automobile tires.¹⁸ The hereditary, wear and tear, and toxic factors in



A



B

arteriosclerosis are paralleled with the effects of poor manufacture, fast driving, and oil on tires. Even the higher susceptibility to blow-outs in such regions of functional strain as the cerebral arteries and the rear tires is pointed out. That such comparisons are more than figurative allusions is shown by demonstrations of decreased elasticity in the aging artery.

Change from aging in elastic tissue fibers is a fundamental structural alteration that leads to widespread secondary morphologic change. In the skin it is the loss of elastic fibers, along with the atrophy of fat, that results in wrinkling. In the lungs senile emphysema follows from the distention of alveoli no longer supported by an adequate framework of elastic fibers. In the arteries it is the degeneration, regeneration, and final replacement of elastic fibers by less functionally adequate collagen that comprises a large part of arterial involution.

When one considers how much of the structural change of senescence, whether primary or secondary, finds its origin in the two fundamental alter-

ations, atrophy and arterial change, the significance of the fact that these biologic phenomena are so closely paralleled by similar reactions in nonliving colloidal systems can hardly be overemphasized. Moreover, it affords at least a beginning for a physical explanation of an even more mysterious phenomenon, namely the rejuvenating effect of cell division, whether of asexual or sexual origin. For it is not a peculiarity of living systems that, newly "mixed," they manifest a high energy content, and that as they age these energies are dissipated. The same is true of the simplest of colloidal systems. Cell division results in the preparation of just such a new system by the rearrangements of its protoplasmic constituents and thus new energies may become available for growth and function.

Other evidence of similarity to colloidal behavior in the production of senescent structural change is seen in the framework of atrophic organs, where the swelling and fusion of collagen fibrils forms a homogeneous hyaline. Since this alteration is commonly found as the accompaniment of cellular atrophy, where dehydration of cell protoplasm is evidenced, a possible explanation of the reciprocal occurrence of these two changes may be found in the shift in water of the colloidal system from the intercellular to the extracellular phase. All such comparisons must perhaps remain as tempting hypotheses until the question has been directly examined in living tissues. The reader is referred to the Harvey Lecture of Hastings for the description of such study.¹⁹

EFFECTS OF CUMULATIVE INJURY AND REPAIR

Much of the effect of the wear and tear of a long life, to which allusion has been made frequently in preceding paragraphs, is of so elusive a nature that it is better described as a "factor" in the production of the structural alteration of age, rather than as the change itself. Such for example is the use of the concept in explaining the localization of arterial change, whether lipoid infiltration or elastic fiber degeneration, in certain parts of the aorta. In other instances the direct result of the usage is the death of the cell and the repair that follows this damage. That the cumulative effect of these replacements must become increasingly significant with the passage of time is evident and their importance in the production of senescent change has therefore received widespread recognition. Theoretical speculation has indeed concerned itself as much with the effects of the repair of these eroding effects of time as with the regressive change itself.

The wearing out and replacement of cells is again a phenomenon that is observed at the very origin of the individual's embryonic life, a senescent change that is well established by the time of his birth. A remarkable variation is noted, however, in different tissues in the rate and degree of replacement of cells. In the bone marrow there is a rapid and constant renewal. In the testis a flood of new cells is produced throughout the period of sexual maturity. In the skin there is also a rapid turnover, with the transformation of undifferentiated cells of the lower epidermis to the dead stratified keratin plates of its surface. So rapid, in fact, is the process that diurnal rhythms of mitotic division have been established.²⁰ At the other extreme is the fixed status of the parenchymatous cellular content of the central nervous system, for apparently there is no replacement whatever of the ganglion cells that may be lost through the course of life. The same seems true of the cardiac muscle fibers, or, at least, replacement of them is very scanty. The glandular organs lie between these two ex-

tremae. Mucous glands and others of the apocrine type where cellular protoplasm is drawn upon for the functional product, show active replacement, but even in a gland which elaborates little in the way of a visible product such as the tubule of the nephron mitotic divisions are frequently enough noted to allow observation of rhythmic activity.

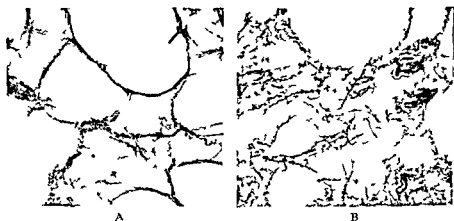


Fig. 24 Age changes in the lungs. The disruption of the atrophic elastic tissue framework in senile emphysema of the lung of sixty-five years (B) contrasts to the thick, well-preserved pattern of the elastic fibers in the lung of eighteen years (A). It will be noted that the degree of strain on the two sets of fibers must have been about equal as the air spaces are equally distended.



Fig. 25 Age changes in the brain. Atrophy and pigmentation of ganglion cells in the deeper layers of the cerebral cortex in a seventy-eight year-old brain (B) as compared to those of an eighteen year-old individual (A). The other prominent lesion of senility, proliferation of glia, is not shown in the figure.

There is much direct experimental evidence that proves regeneration and

cells do proliferate in replacement are often atypical in both structure and function. So striking is this alteration that in many instances islands of metaplastic tissue stand out on the background of normal tissue type. Patches of metaplastic stratified epithelium scattered along the normally ciliated columnar epithelium of the trachea and bronchi and in the transitional epithelium of the renal pelvis are so interpreted. Other evidences of difficulty in replacement that occur with aging are seen ultimately even in tissues of such active cell exchange as the skin and the testis, for the years eventually bring a slowing of division and atrophy, with decrease in size and differential complexity of the constituent cells.

In certain instances the cumulative effect of years of continued breakdown with a repair which does not replace *exactly* the original structure of the tissue leads to widespread change in the original architecture of the organ. The old kidney or artery is not merely a simpler and smaller organ, but one built *differently*, and the functional disturbances that result from this trans-



Fig. 26 Age changes in the skin. The most striking change in the seventy three-year old skin (chest) (B) is the disappearance of the black-stained elastic fibers from the deeper layers of the dermis as compared to the great number seen in the twenty two year example (A) from the same region. There is also atrophy and disappearance of the papilla, though the epidermis shows no thinning in this example.

formation are not therefore a simple inadequacy, but an alteration of the original function. The pathologist Jores²¹ has suggested the word *metallaxis*, a term that contains not only the connotation of rebuilding but also of transformation, for these architectural alterations.

The *clinical implications* of this concept are evident, though perhaps not altogether sufficiently appreciated. Some of the mechanical disturbances that result from the transformation of the arterial wall are easily understood, such

nicely balanced composite of elastic tissue and spiral smooth muscle²² are less clear, although they doubtless are responsible for many obscure clinical evidences of circulatory difficulty.

In a complex organ such as the kidney even greater functional disturbance may follow architectural transformations of nephrons. It has been shown in

old dogs that the atypical renal epithelium which lines the proximal convolution has lost its functional ability to take up and concentrate within its cells certain foreign substances. The renal mechanisms for this substance, trypan blue, have therefore been entirely changed from the normal manner of handling the dye.²³ What effect this replacement by a relatively functionless epithelium may have on the handling of physiologic substances by the nephron we cannot tell until the specific data regarding them is obtained. However we may at least suspect that the general clinical application of physiologic tests to the diseased kidney should be subjected to rigorous criticism, for all the evidence that we do have in the matter suggests that the interpretation of "clearances" may be quite different in a kidney which is built and which functions differently from the normal one, on which our knowledge of the "clearance" is based.

As has been stated, considerable theoretical importance has been attributed to the *proliferative and regenerative aspect of senescent change*

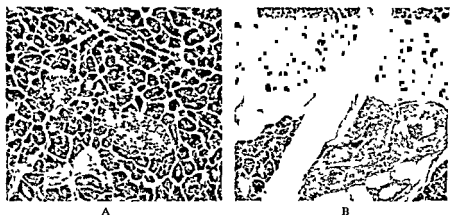


Fig. 27 Age changes in the pancreas. Some decrease in size of both secretory acini and islands of Langerhans is seen in the seventy five year-old organ (B) as compared to that of twenty years (A). The latter are commonly reduced in total number in old age. More striking however, is the great thickening of the arteries and the infiltration of the framework of the organ with fat.

Much of this theory stems from the work of the biologists Calkins and Childs and the embryologist Minot. The former, studying unicellular organisms, observed definite evidences of strain rejuvenation resulting from not only sexual but also asexual division. Minot's contribution was that an extreme of cellular differentiation leads to a dead-end of senescence. This last can be reversed by cellular division and a consequent dedifferentiation to a simpler structural form, thus opening the way to advancing developmental progress. The leap

rejuvenated, but in fact ages and becomes functionless more rapidly than does the cerebral cortex, where no division whatever occurs in the ganglion cells.

The increased incidence of tumors in old animals has also been ascribed

to the regenerative disturbances that occur in senescent tissues, and with much stronger evidence. Goodpasture's²⁴ study of anatomic changes in old dogs led to the conclusion that "from these dedifferentiated cells metaplasia and benign and malignant tumors arise," a statement which does not preclude the importance of other factors in the genesis of neoplastic growth in the aged. There can be little doubt, for example, that epitheliomata of the renal pelvis, and probably those of the bronchial mucosa, have their origin in some such relation of metaplasia to anaplasia.

Further effects of metaplastic or atypical replacement are seen in MacNider's demonstration that the atypical regenerated epithelium in the liver and kidneys of dogs is less susceptible to damage by a second administration of uranium nitrate, chloroform, or ether than the original epithelium which has been replaced.²⁵ This atypical regeneration is more pronounced in old dogs and the escape of the cells from the second toxic effect seems linked to



Fig. 28 Age changes in the thymus. The thymus of thirty years (B) shows extensive involution as compared to the organ of sixteen years (A). There is widespread atrophy and disappearance of lymphadenoid tissue, degeneration and calcification of Hassall bodies and diffuse interstitial fibrosis. There is also extensive thickening of the arteries of the involuted organ, though the arterial system elsewhere was well preserved.

their atypical structure, for regenerated cells of normal type in young animals succumb. MacNider has also shown that chemical factors, such as shifts in the acid-base equilibrium (and these are known to occur more readily in old age), are involved in the susceptibility of the kidney and liver to damage with these substances, so that the exact mechanism of the "immunity" is not yet clearly understood. It may be that the inability of atypical, relatively functionless cells to take up and concentrate within themselves the toxic foreign substance, similar to their demonstrated inability to take up and store trypan blue, is the reason for their escape, for it is known that most of the original epithelium of the normal nephron does not concentrate foreign substances and is therefore never affected by renal poisons.

HORMONAL CONTROL AS THE INTEGRATOR OF THE STRUCTURAL CHANGES OF SENESCENCE

In the preceding pages various structural changes that result from the passage of time have been described as isolated phenomena. As they are ob-

served in actuality, all are found in varying degrees and combination in one individual, and his senescence, which is peculiarly and characteristically his own, is the sum total of their effects

That senescence is a biologic phenomenon of constant occurrence, and indeed the simple fact that we can describe it at all, indicate that it is an orderly if variable process. Consequently some mechanism must exist that is responsible for the integration of the complex interrelations between the various structural changes

It would seem likely that the mechanism concerned in the integration of the normal involutions of late life must be one or both of the two that have operated during the course of the individual's earlier existence and that their influence continues up to and through the gradual appearance of senescence. There is little evidence of nervous integration of the structural changes of old age, whatever part it may play in the correlation of its functional disturbances. There is much to suggest, however, that the complex and widespread changes

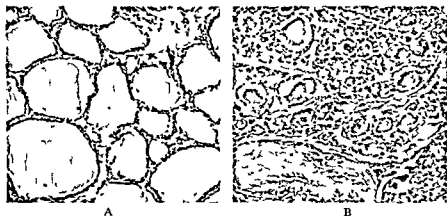


Fig. 29 Age changes in the thyroid. Atrophy of follicles with scanty colloid and heavy interstitial fibrosis in the seventy year-old organ (B). There is also an extreme sclerosis of the arteries to the organ. The contrasting figure shows the organ at twenty five years (A)

in structure throughout old tissues and organs are correlated by the chemical influence of the endocrines. The experimental studies of Leo Loeb⁸ on the aging of carefully established strains of mice have done much both to elucidate nebulous general theory concerning the role of hormones in the process of aging and in indicating how their action on living tissues 'functions approximately as a harmonious system, but becomes more and more disequilibrated with advancing age'. Our interest is particularly concerned with this study, since it was the structural effects of hormonal control that he examined in such organs as the thyroid, ovary, adrenal, and skeleton.

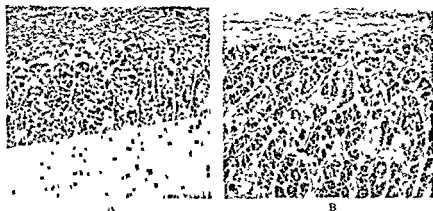
The structural changes of old age were first carefully established in a

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at the epiphyseal lines with later calcification and degenerative changes of

sclerosis and absorption were prominent. These changes differed in both the time of occurrence and the degree of their evolution in different strains of mice. The constancy of these variations indicates that the basic structural changes of senescence are fixed and hereditarily determined strain characteristics. To quote Loeb directly: "They are not produced by environmental factors . . . of which the action of hormones are an important constituent." The importance of this fundamental statement will become apparent in the discussion of the so-called unitarian theories of senescence that follows.

That hormones do influence the development of the structural changes of aging is certain. Loeb's experimental analysis of their effects shows how the same substance, acting on different organs and tissues, may, depending on the time, the manner, and the degree of its administration, produce effects of strikingly different variety in organs as widely apart as the uterus and the skeleton. Estrogen accelerates and intensifies the structural changes of age in both. Anterior pituitary transplants loosen the dense stroma and produce a



46. 30 Age changes in the adrenal gland. The rows of cortical cells of the seventy-two year-old organ (B) are narrowed due to the atrophy of the cells and there is an accompanying increase in the delicate connective tissue framework which contrasts with plump cords and scanty framework of the organ at twenty-two years (A).

sort of rejuvenation in the thyroid and mammary glands, while on the epiphyseal cartilage they produce at first youthful proliferative change followed by the senescent changes of degeneration and absorption.

To summarize, in Loeb's words, the part played by hormones in the correlation and integration of the widespread alterations of senescence: "Their action is not restricted to a single organ or tissue so much as was formerly believed." From this general regulation comes much of the specific pattern that senescence presents in the individual. "We see then that hormones are able to accelerate or intensify some old age changes and to retard or even reverse certain others, and that the same hormone may induce both these effects under varying conditions. But it has also become evident that these effects of hormones are limited by the inherited constitution of the living substratum, and that they cannot accomplish much more than to spur on or to inhibit tendencies which are performed in the substratum on which they act."

THE STRUCTURAL BASIS OF UNITARIAN THEORIES OF SENESECE

A consideration of the theoretic aspects of the structural changes of senescence must at least briefly consider those not infrequent claims that find in some single factor the origin of the senescent process. Often this localization is remarkably exact, in a specific organ, and, as if this organ had been a sort of fountain of youth, all the phenomena of aging are drawn from

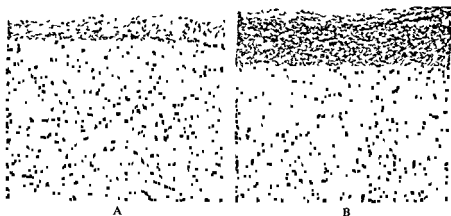
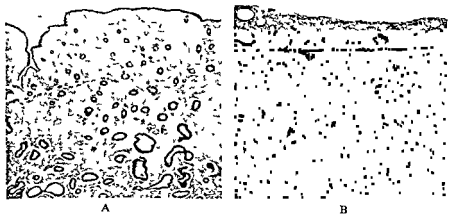


Fig 31 Age changes in the ovary. In the sixty-year old ovary (B) no ova remain and the stroma of the organ is dense and more fibrous than that of the corresponding organ at eighteen years (A)



failure of its source. The final argument in such reasoning is the demonstration of some definite structural alteration in the organ which serves as the fundament of the whole theoretical elaboration.

Endocrine System. It is among the endocrines is particular that most of these prime movers of senescence have been sought. In the light of Loeb's studies, no farther extended consideration is necessary of the Brown-Sequard

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theory of sexual atrophy as a reversible mechanism of aging or of its derivative applications such as the rejuvenation technic of Steinbach unless it is to note that with the recent synthesis of gonadal hormones there arose in some an immediate although quickly subsiding hope that youth might now be administered hypodermically

Other similar speculations are less obviously fantastic though fundamentally no less inadequate. For example the suggestion that senescence is hypothyroidism with its lowered metabolic rate, muscular weakness, wrinkled skin and sparse hair. When the thyroid of the aged is found in fact to be atrophied, all is apparently answered except that one can still ask if the structural change in the organ might not as well be an effect of aging as its cause. Similar sequences have been or could be built around the adrenal, the pituitary and every other endocrine organ.



Fig. 33 Age changes in the testis. There is an extreme interstitial fibrosis with so great thickening of the basement membrane of the tubules in the seventy-two-year-old testis (B) that the lumen of the seminiferous tubules is greatly reduced. Contrast the shrunken atrophied spermatogenic cells with the pyknotic nuclei and the absence of free spermatozoa with the proliferative activity in the tubules of the twenty-year testis (A). Between the tubules of the senile organ are seen persisting interstitial cells.

Central Nervous System Considerably more refined are the theories of Ribbert²⁶ and Mühlmann²⁷ that locate the essential change of senescence in the central nervous system. Not that other factors and evidences of the gnawing tooth of time are denied, but a natural death from old age is said to be the result of structural change in the central nervous system. Atrophy of the ganglion cells of the cerebrum, with pigment and lipid accumulation, is the structural cause of impeded function that progresses slowly and inexorably to a gradual cessation of consciousness and final extinction. Even the most enthusiastic supporters of the theory admit that rarely ever does an individual survive the other and presumably unnatural effects of the years long enough to enjoy this ideal exitus.

Arterial System The statement of another theory of senescence, that a man is as old as his arteries, errs perhaps more through its epigrammatic terseness than from inherent fallacy of its claims, as there can be no question that many of the disintegrations of old age are directly due to the effect of vascular change. The example of the old human kidney has been cited as an

organ where there seems little need to assume any other cause for the alterations of aging. It is moreover also true that the general ill effects of generalized vascular senescence are in part not the result of the change in arterial walls but of an increased tension within them (See p 468)

Summary. For all these simplifications of the complexity of aging there can admittedly be found a structural basis of some sort in tissue change from which theoretical elaboration can proceed. It is also certain that all these effects contribute to the establishment of senescence. The error in them arises from the fact that it is impossible in the face of an almost infinite multiplicity of causes and effects to trace a direct chain of events without ignoring a large part of the available data. Attempts to establish some as "primary" and others as "secondary" only complicate the difficulties and lead to controversy over relative values. What is needed therefore is a reasonable way of looking at the problem.

STRUCTURAL CHANGES AS CONSTELLATIONAL FACTORS IN SENESCENCE

If one considers senescence as a general concept, including all the complex phenomena observed in any aged individual, then no better example can be found to illustrate the value of what the pathologist Tendeloo has described as the method of "constellational pathology."²⁸ According to this manner of reasoning it is futile to seek for *the* cause in even the simplest of pathologic phenomena. Rather there are concerned multiple causes which operating in some specific orientation of both time and space, constitute a "constellation" that determines the particular pathologic condition. In the simpler phenomena many of the factors may be known, but some always escape our knowledge. Hence the apparently exceptional or atypical reaction so commonly seen by those whose daily experience is concerned not with the ideal description of the textbook, but with the actuality of practice, either in the laboratory or clinic.

A relatively simple example of the application of this reasoning may be seen in a consideration of the meningococcus as the "cause" of the purulent meningitis that usually, but not always, is the prominent structural lesion of epidemic cerebrospinal fever. That the organism is not the determining cause is shown by the occasional case where, the bacteria reaching all tissues, including the meninges, no meningitis develops, but death results from septicemia.

bacteria, of various localizing effects of the tissue and tissue fluids, of the reactivity of the tissue cells, of balance of ferment and anti-ferment, all of these, no single one, by their relation to each other acting as a constellational cause to determine the end result.

How infinitely more complicated is the problem of senescence in which one must consider the whole organism reacting to the summation of all its contacts with a changing environment over the course of years. The known factors that correlate to produce the aging effect are legion and those still

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the choice is utter incomprehension or the adoption of the one-tracked

hypothesis that ignores all but the convenient. Our previous consideration of such unitarian theories has shown the futility of this latter course.

Even in the state of our present ignorance some order can be achieved by Tendeloo's method, and its admission that there are still missing factors in the constellation allows room for growth in understanding. For example, much has been said of the structural change of brown atrophy, and its occurrence in the heart muscle cells has been described. Yet it is common knowledge that the weight of the heart usually increases with aging. Equally familiar is the occasional senile heart that is definitely small, particularly in individuals who live to great age. Is the pigmented atrophy of the cells and the increased weight of the organ a factual discrepancy and are we forced to the feeble admission that the occasional small heart is "the exception that proves the rule"?

Many of the factors involved in determining the size of the heart in old age are known. There are two factors that directly determine the size of the



A

B

Fig. 34 Age changes in the prostate. The uncomplicated senescent change of atrophy, not hypertrophy, is seen in the shrunken glands of the seventy-eight year organ (B). The interstitial tissue is at least relatively increased in amount. Note not only the large size of the gland acini in the twenty-two-year-old organ (A) but also the high epithelial cells which line them.

muscle cell. Sixty-four years of age. The same tissue as in Fig. 33, but at a lower magnification.

individuals either early or late in life. The first factor, largely determined by the pressure in the arterial system against which the blood must be moved, increases slowly in the later years. Recent evidence shows that the degree to which the kidney has aged may be at least in part a factor responsible for this mild hypertension. But aging of the kidney again is largely the result of aging of its arteries and this arterial senescence is a part of a general process in which uncertain factors may produce localization in the renal vessels by what appears at present to be fortuitous chance. Moreover, there is evidence that the effect of the renal "ischemia" acts by humoral mechanisms both directly on the arteries and also through the central nervous system. And it is further certain that senescent structural change in the peripheral arteries, such as fibrosis, medial hypertrophy, and reduction of lumen, must be factors that

modify the functional response of these vessels to this control. And so one might continue.

It would be folly in the state of our present knowledge to attempt to set up formally the constellation of causes that determines the size of the heart in old age, but enough is known to recognize that this method of considering the complex phenomena of senescent change leads at least to a glimmer of understanding, where the old concept of simple cause and effect leaves us distracted by multiplicity of data and contradiction. We can understand why a senile heart may reasonably be either large or small and can search, at times with success, for the structural evidences of its constellational cause in arterial thickening, renal scarring, and cardiac size.

A BRIEF RÉSUMÉ OF ORGAN ARCHITECTURAL CHANGE IN SENESENCE

A factual description of the alterations of organ structure in senescence that will fit into the confines of this chapter presents considerable difficulty in exposition, for unless specific detail of cellular change and rearrangement is fully presented, a briefer statement of essential changes that are common to all the tissues gives no picture of the individual senile organ. The spleen, brain, and heart must be described as showing atrophy and disappearance of their more highly differentiated cells, more or less pigmentation, interstitial fibrosis, and vascular sclerosis. To avoid this difficulty the changes in the various organs are therefore presented in graphic form. For fuller detail of histologic description than is found in the legends of the figures, the reader may refer to Cowdry's "Problems of Ageing," where representative articles describing the changes in each organ are given.³

Figure 35 shows the weights of important organs from birth to senility. The graphs serve several purposes.

First, the factual evidence of the waxing and final wane of each organ is presented.

Second, it will be noticed that there is great variation in the degree of change in different systems. The female gonads are the most labile of all the organs, the ovary presenting the most striking peak at sexual maturity and the greatest fall in old age. It is interesting to contrast with this the more even maintenance of weight in the senile testicle. Another female sexual organ, the uterus, shows a similar but lesser decline in old age. The viscera, spleen, heart, liver, and kidney show a definite decrease in weight in late life, while certain endocrines, the thyroid, adrenal, and pancreas, as well as the brain, maintain their mature weight more constantly.

Third, the curve of weight change in any organ may be taken as rough evidence of the degree of senescent structural alteration that occurs in the organ. This is true, because one of the most pronounced of these alterations is atrophy of parenchyma, though when there is a large amount of interstitial stroma, as in the brain with its extensive glial framework, considerable change is possible without much loss of weight.

With the distribution of senile structural change throughout the body roughly indicated by this graphic method, the architectural arrangement of

and different organs may be seen on Figs. 20 to 34. Each pair

Concept of Age. It is universal custom to define the age of the individual in years, and this rough measure has been followed in the selection of our series. In considering these examples of senescent change it must be remembered, however, that the age in years given for each specimen has little absolute significance, for chronologic age and physiologic age, both functional and

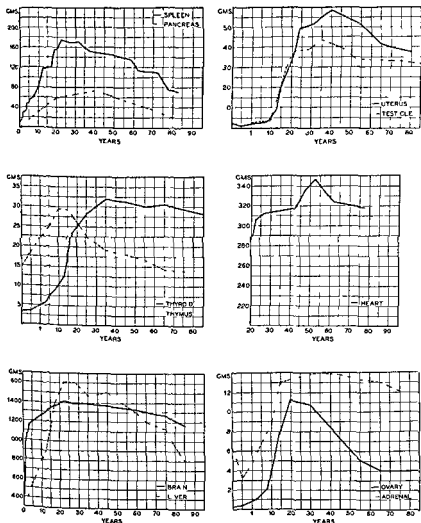


Fig 35 Rise and fall of weight in various organs with the passage of time (After Roessle and Roulet ²²)

structural, as even the layman well knows, do not run concurrently. Examples showing the same degree of structural alteration might have been chosen from individuals ten years or more older or younger than the one which appears in our series as an illustration of senescent change.

A more valuable scale for the determination of the age of a man could be established on the degree of senescent change that had occurred in the in-

dividual as a reaction to his environment. Life periods might be set up on the basis of the structural changes that had so developed and these correlated with their functional characteristics. The establishment of such a scale of aging will doubtless be one of the next steps in the development of our knowledge of aging and will be a valuable quantitative scale for the clinician who is interested not in the years of a man but in the man that he is.

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CHAPTER 5

MENTAL CHANGES WITH NORMAL AGING

WALTER R. MILES AND CATIARINE C. MILES

PSYCHOLOGIC aging is observed by everyone. The young regard the process as remote and of no immediate concern. Those effective in middle age attempt to postpone its recognition in themselves, while often well aware of it in others. Those in late maturity who are competent for their age accept the changes with good or poor grace, thankful that they are not yet senile. The very old, where they recognize the changes, prefer to formulate them in physiologic or anatomic terms, or if they must, in specific rather than general psychologic ones. Thus they are willing to recognize failing vision or lessened physical strength, fatigability, or even absent mindedness. The aging person quite naturally does not want to admit that the total psychologic organism, the personality, is changing in an unfavorable way. Nor should he, because until the true course of adult personality development is plotted in detail with as full regard for positive as for negative aspects, the available picture, although correct as far as it goes, may be fundamentally misleading. At the

edge and experience and on the seasoned traits of maturely organized emotions and life objectives resists objective appraisal. For the physician in practice, acquaintance with the normal outlines and perspectives of the changing mental picture, as far as they have been traced, affords a background against which individual aging can be appraised as approximately normal or as distinctly pathologic.^{1,2}

Human engineers, including physicians, having undertaken the servicing of their fellow human beings, have assumed the responsibility of knowing what normal life development is, not only in childhood and in late age, but also throughout the period of the middle adult years. The growth of the mind and the development of the personality in childhood and youth have been the subjects of psychologic study for a half century. Progress has been most rapid in this field in the twenty five years just past. And in this same period a beginning has been made in drawing upon the techniques and concepts found useful in the study of development in the early years for the measurement and appraisal of changes in earlier and later maturity.³ At the same time there has emerged a new emphasis on life experience as a dynamic expression of each individual, on the personality as a whole as the ultimate object of psychologic study and evaluation. This emphasis calls for a balanced picture in which each of the complex elements in the living person shall be appropriately regarded

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dividual as a reaction to his environment. Life periods might be set up on the basis of the structural changes that had so developed and these correlated with their functional characteristics. The establishment of such a scale of aging will doubtless be one of the next steps in the development of our knowledge of aging and will be a valuable quantitative scale for the clinician who is interested not in the years of a man but in the man that he is.

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PSYCHOLOGIC aging is observed by everyone. The young regard the process as remote and of no immediate concern. Those effective in middle age attempt to postpone its recognition in themselves, while often well aware of it in others. Those in late maturity who are competent for their age accept the changes with good or poor grace, thankful that they are not yet senile. The very old, when asked to appraise their own mental changes, regard them as physiologic or anatomic rather than as psychologic ones. The young, with their physical strength, quite naturally does not want to admit that the total psychologic process is

present time decreasing function expressed in rate and quantity of accomplishment, in plasticity, and in originality is readily amenable to measurement;

which individual aging can be appraised as approximately normal or as distinctly pathologic.^{1,2}

Human engineers, including physicians, having undertaken the servicing of their fellow human beings, have assumed the responsibility of knowing what normal life development is, not only in childhood and in late age, but also throughout the period of the middle adult years. The growth of the mind and the development of the personality in childhood and youth have been the subjects of psychologic study for a half century. Progress has been most rapid in this field in the twenty-five years just past. And in this same period a beginning has been made in drawing upon the technics and concepts found useful in the study of development in the early years for the measurement and appraisal of changes in earlier and later maturity.³ At the same time there has emerged a new emphasis on life experience as a dynamic expression of each individual, on the personality as a whole as the ultimate object of psychologic study and evaluation. This emphasis calls for a balanced picture in which each of the component elements of the total psychologic process is regarded

associated.

the individual as a whole. Mind is viewed as a function of body and it is generally recognized that bodily behavior offers means for observing mental activity. Behavior completely viewed is personality in function. Cross-sectional behavior studies at significant points in the life span of groups afford outlines of reference or norms. Schematically viewed, such series can be thought of as presenting longitudinal graphs of typical phases in life development. Ideally the individual should be viewed always with reference to his place in an infinite number of trait dimensions. In practice most of these are not available.

THE PSYCHOLOGIC OUTLINE

In the development of modern psychology, a working scheme based on practical experience has gradually emerged as a framework for study of the psychophysiologic organism, the human person. This includes the outward appearance and physique of the individual, the sensory and motor functions upon which ability and performance depend, attention and memory with their intellectual product, learning, intelligence or mental capacity, the chief determiner of intellectual achievement, emotional attitudes and motivations, and finally, interests, occupations, and productions, the behavior molds into which individual mental traits, trends, dynamics, and powers are formed.

Appearance and physique carry a psychologic as well as a physiologic record of age. They depend on life experience and mood as well as on physiologic and anatomic structure. The subjective feeling of youth or of age influences their status, and in turn their preservation affects the total course of psychologic aging. The characteristic decade to decade changes in the facial and bodily expression are themselves an index of age. These are discussed elsewhere in their physiologic setting. It is sufficient here to mention their place in the psychologic picture.

SENSORY AND MOTOR ACTIVITY

Sensory and motor functions, like appearance and physique, are correlated with chronologic age throughout the life span, in childhood positively, in adult life negatively. Of the sensory functions, hearing and vision have been most often and most carefully studied because so large a proportion of experience is gained primarily through them, and because they are readily measured.

Hearing. Hearing shows a well known gradual loss with age, noted first in the higher tones (See Chapter 20). At 8192 cycles the age curve from twenty to sixty years drops about 40 decibels. At age forty the decrement

of age with highest audible pitch is negative, about 5. Hearing loss may interfere with social activities, with the enjoyment of music, and with effectiveness in work relationships. Generally, within the degrees of normal loss occurring up to the seventh decade, individuals are able to compensate by attentiveness, thoughtful observation, and adjustment of position for what the

measure of physiologic age (See Chapter 19). Visual acuity, a prime essential

for psychologic experience and activity, in its decline also gives a basic index for psychophysiologic aging—the well-known presbyopia measurement. Comparisons of the mental imagery of blind persons who formerly had sight with the blind who have never seen show that the cumulative effect of long visual experience prepares those who in late age meet a dimmed environment. Furthermore, mechanical correction of vision through glasses enables most normally sighted adults to continue throughout maturity with those activities depending on vision which are important in social life and in occupational activities.

Visual perception offers perhaps the chief means of sensory contact with the environment in the experience of normal people. It depends on visual acuity, which perhaps need not be better than 20/50, but is itself a central psychologic function. Reading speed and comprehension, the viewing and interpreting of the physical environment, depend on visual perception. This function measured with short time exposures of various types of materials shows correlation coefficients with age between $- .43$ and $- .56$. Important is the finding that decline is relatively less for individuals who have characteristically made greater use of visual materials. Looking at motion pictures and recalling their content is near enough to true life experience for tests so made to give a fairly reliable measure of typical visual memory experience. In a population of more than 600 persons ranging in age from the twenties to the fifties, decrement appeared in each successive age group. The loss was in the specific function of *visual memory* and was found to be unrelated to speed of reaction, motivation, visual acuity, or amount of education.⁵

Visual perception and visual-motor coordination of highly specialized kinds are basic in many occupations. Tests designed to measure the finer perception-response behavior of experienced workers show regular age decrement in adult competence. For example, perception work-rate has been studied in a standard test performance with groups of skilled seamstresses. The younger workers, aged twenty one to forty-one, accomplished definitely more in a long test series and made fewer errors than old workers, aged sixty-seven to eighty two. The old workers showed an interesting favorable characteristic, however. They maintained throughout the long test series a more even and consistent work level than the younger women, with a rate that was never less than 94 per cent of their best record.

Motor Ability. Motor ability is basic to activity and activity is generally essential to normal living. Hence the measurement of the correlation between motor ability and age gives one index to activity in work competence and recreational adjustment in maturity. Correlations of a number of elementary motor abilities show in Galton's large populations (upwards of 3700 persons aged twenty five to eighty-one) coefficients ranging from $- .25$ to $- .34$. The abilities measured include hand grip, strength of pull, and swiftness of blow. In all of these functions decline of ability is progressive, beginning at twenty

sound. Correlations for populations of 400 to upwards of 800 persons show

more experienced functions. Habits established through exercise although sometimes spoken of as associated with the rigidity of old age, have thus their favorable aspect in the better maintenance of experienced effort.

MANUAL SKILL

Tests of Manual Skill. In complex manual activities, the characteristic speed or dexterity loss regularly appears as age advances. Miles, using the Link McFarlane Cube Assembly Test, a measure of perception, dexterity in construction, and intelligence, with a representative population of adults from twenty to ninety years of age, found gradual slackening in speed from the twenties to the sixties, with a sharper decline in rate thereafter. That practice and experience contributed to speed of performance was shown by the work of men with definite mechanical training. These maintained the young adult speed rate into late maturity and were significantly superior in performance in the age group fifty to sixty-nine to mechanically untrained men of equal intelligence and a similar amount of formal schooling. Another test of manual skill (McFarlane Coat Assembly Test) reported by Miles for a population of more than 800 adults shows again the speed age-decrement (see Fig. 36), and here the experience or practice factor entered in markedly to retard the age influence. Both men and women were slower in old age than in young adulthood, but the experienced group (in this case the women) made practically as fast a record in the forties and fifties as in the twenties and thirties, they were a little slower in the sixties and seventies, and thereafter declined rapidly. The men's speed loss was gradual from decade to decade and was, in all, twice that of the women from the twenties to the eighties. Typical experience resulting in trained skill gave the women in every decade a work rate significantly faster than the men, even at seventy the average woman was better on this test than the average twenty-year-old man. A group of men tailors, however, equalled the women's speed score, thus corroborating the interpretation of experienced skill as a delaying factor in speed decline.

Motor vehicle driving is itself a test of manual skill, depending on visual perception and, obviously, also on judgment. Three essential psychophysiologic elements in driving efficiency are glare vision, steering coordination, and braking reaction time. Measurements of these functions by age groups follow other age curves of skill in maturity. Each of the three shows decline of skill with age, but this occurs in varying degree. Sensitivity to glare, a basic sensory function, shows fairly rapid decline in efficiency after reaching its peak level near age thirty. Braking reaction, a motor speed function, reaches its peak at twenty-five, declining fairly rapidly thereafter. Steering, a complex eye-hand coordination skill, also declines from a peak at twenty but the curve is more gradual and at forty-five less loss has been sustained in this than in either of the other functions. These results, when regarded in the light of

... prefer slower open-road speed is no doubt one element in their safer driving. Measured in mileage per fatality, all ages from thirty to sixty-five are

superior to the twenties and far superior to the especially hazardous teens. The peak of safety in mileage terms is in the late forties when greater experience and a heavier sense of responsibility bring greater activity and maintain greater competence in spite of the considerable sensory and motor decrements that have already occurred. DeSilva,⁶ who reports these results finds that his composite "good driving" scale shows an upward curve of ex

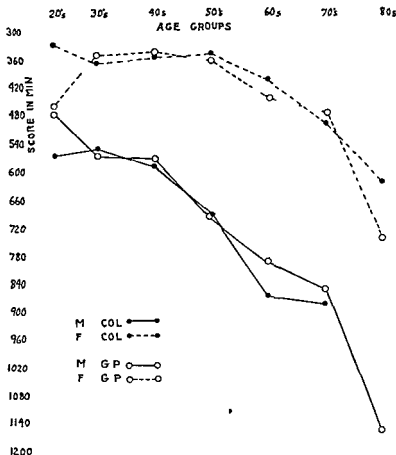


Fig 36 Decline of speed with adult age in a test of dexterity and skill (McFarlane Coat Assembly Test) (Miles in Murchison's A Handbook of Social Psychology Clark University Press) Male and female populations sampled represent the general public and college educated

perience with age from thirty four to forty two, improvement in a highly practical sense outweighing specific age loss in function

A unique study on aging in man and his capacity for *physical work* deserves mention in this chapter.⁷ It was made on the same man at ages 41, 53, 57, 68, and 71 years. The work performed was riding a bicycle ergometer provided with a cyclometer and adjustable mechanical or electrical brake. Prior to each of the test periods at the different ages the subject attempted to achieve prime physical condition through walking, mountain climbing, and

running At age 41 the subject's top bicycle-ergometer work output in a 30 minute period was 55,000 kg m, that is, 1830 kg m per minute In comparison with this, the 30 minute work tests at succeeding ages gave per minute kg m values as follows age 53 1051, age 57 1089, age 68 850, and age 71 953 In summary, it was found that the working capacity declined with age, reaching at 71 years about 50 per cent of what it had been at age 41, the work score was about 3.5 per cent higher in the afternoon as compared with the morning at ages 57 and 68, and the number of days required to recover from a work test was found greater at ages 68 and 71 than at age 57

INTELLIGENCE AND AGE

Results from intelligence tests by many investigators give clear-cut evidence of (1) score decline from young adulthood to old age, (2) greater decrement of the speed versus the "power" of intelligence, (3) better preservation with age of the verbal as compared to the mathematical and manual functions, (4) wide individual differences in score at every age Investigators in different localities and with diverse populations, representing similarity and dissimilarity with respect to economic, occupational, social, and educational status, report results in substantial agreement with respect to score decline with age in adulthood from a peak generally in the late teens or early twenties The parabolic curve sometimes shows a sharper drop in extreme age (seventies or eighties)

The age score curves are similar to those for other psychophysiologic functions, including sensory and motor efficiency and more complex skills, and indicate here a similar hierarchy of greater and lesser decrement If speed of reaction or youthful vigor is essentially involved in the test performance, decrement begins to appear early in adulthood, is continuous and tends, in later age, to become excessive The correlations between score and age in speed tests of intelligence range from $- .45$ to $- .55$ for the span of maturity, ages twenty to ninety five ^{8,9} Miles and Miles, reporting on more than 2000 cases, found a correlation of approximately $- .50 \pm .02$ for this age range A number of investigators, reporting on groups measured in early and middle maturity, found correlation coefficients for ages twenty to sixty-five amounting to approximately $- .30$, from fifty to ninety-five years the coefficients are near $- .40$ In speed of intelligence, adults generally suffer a loss from decade to decade amounting to approximately one quarter of the standard deviation of the young adult The decade score loss of the average person on speed tests amounts approximately to four and five tenths mental age months per semi-decade This means an average loss of 3 IQ points per decade Wechsler's careful standardization of an adult individual intelligence test of the Binet type shows similar results, and his report and our own observations of the decrement found age by age with the Stanford-Binet Scale indicate that the average person suffers an equal loss with age in terms of this test ¹⁰

On untimed or "power" tests of intelligence, score decrement occurs with age, but the rate of decline is slower With the Otis higher intelligence test (B, 75 items) and with a shorter form of the same test (thirty-six items, numbers 40-75 inclusive twenty to ninety with a group of 143 individual CAVD Tests ¹¹ The average decade scores of men and women are generally

about equal whatever test is used, and they describe approximately the same decrement curve

Tests of intelligence are generally made up of a considerable variety of item types. Thus the historic Army Alpha includes oral directions, arithmetic problems, practical reasoning, vocabulary, disarranged sentences, number series completion, verbal analogies and general information. Test correlates closely with test (84+) in representative young adult age groups, and there is no test which does not show score decrement with age when adults are measured decade by decade from early to late maturity. Decline with age

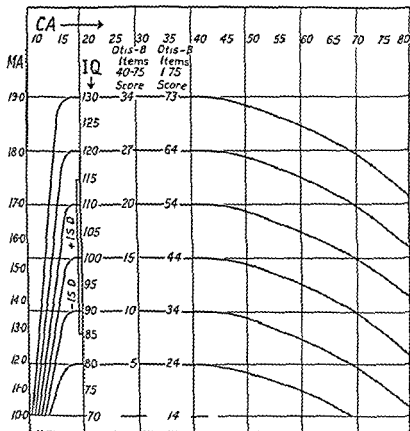


Fig 37 Smoothed curves indicating the influence of age on intelligence test scores of older adults

varies for different types of material, however. Thus, Miles has reported that an information test of intelligence (Terman-Miles) shows increment rather than decrement up to the late forties, with decline after age fifty. Investigators are agreed that among the types of items included in most batteries a hierarchy exists from the verbal items through reasoning to the mathematical and performance types. In "power" tests we have found correlations very close to zero (0 to -.09) in word meaning, synonym antonym, and multiple choice vocabulary identifications. Logical relations and syllogisms suffer slightly greater decrement in nonspeed tests (-.10 to -.15). Arithmetic computation

and reasoning and number series completion tests show larger decrement, registering in power tests score-age correlations of -20 to -30 . Wechsler's results and those of Weisenburg and his associates indicate that performance tests show score decrements with age as large as timed verbal tests (-35 to -53)¹²

Range of Individual Score Differences. At every age individual score differences are far larger than average decade to decade losses. The range from the lowest to the highest of the middle 50 per cent of the young adult population includes from IQ 90 to IQ 110. At every adult age the best and the poorest-scoring 25 per cent differ from the average by an amount that is more than three times the usual decade to decade loss. *In the fifties the best scoring 25 per cent exceeds in intelligence speed more than 66 per cent of young adults. In the seventies the best quarter exceeds approximately 40 per cent of young men at the peak age.* Sward, in a study of forty-five university professors 60 to 80 years old in comparison with forty-five academic men of ages 25 to 35, found that individual differences were larger than age differences, even though the scores in six of his eight tests yielded a significant difference in favor of the younger men.¹³ Tests calibrated in adult age terms, notably Wechsler's Bellevue Scale, preserve the relative IQ relationship from age to age. With other tests age corrections can be made when the constant rate of score decline is known. Differential losses (*i.e.*, deviations from the expected norms) in terms of types of function serve in clinical studies as symptomatic indicators of pathologic conditions.¹⁴

AGE AND LEARNING

Experimental psychologic results are in agreement in indicating age decrement in adult learning. At the same time they as definitely support the formulation that no one is ever too old to learn. In childhood active, varied learning is the rule, in maturity active learning is usually practiced in areas defined in terms of interest. The measured rate of learning follows the characteristic parabolic curve of rapid rise in childhood and youth to a young adult peak, with a slow decline thereafter to late age. In laboratory experiments correlation coefficients like those in other mental functions show the characteristic gradual score decrement with age. Thorndike's studies indicate that adults in the forties learn approximately 85 to 95 per cent as efficiently as twenty-year-olds.¹⁵

Measurement of Adult Learning. Adult learning has been measured in simple sensorimotor tasks and in verbal and mathematical habit formations more or less complex in character. Whatever his age, the learning individual improves with training. There is no veto power to learning exercised by age at any period in the normal life span. Relative decrement occurs, it is true, but in amount this is less than the difference in rate between moderately fast and moderately slow learners of equal age.

There is a high degree of correlation between the rate of learning and differences in

ally follows interest, however, the waning of capacity is retarded by the favorable factors which motivate the choice of the material to be learned

For many groups of young adults from the twenties to the forties, Thorndike's correlations between age and amount learned range from -02 to -22 , when intelligence and initial score in the subject to be learned are held constant. For the same groups of learners, intelligence and initial proficiency in the subject to be learned decline more with age than do the speed and effectiveness of the new learning. The age-intelligence relationship is -29 , age-proficiency, -16 , whereas the median value indicating the age learning relationship is -08 . *Experiments comparing simple and complex learning* reported by Ruch show that decrement increases with complexity when the tasks are not of the learners' own choosing.¹⁶

Factors in Learning. Learning obviously depends on attention, retention, and recall. That these separate elements show greater decline with age than does learning itself means that in the latter other factors are also involved. We have mentioned interest as one of these. Attitude is another. The correlation index of age and attitude with intelligence constant has been found to be $+12$ among younger adults (to age forty). Series of correlations between attitude and age are among those that show a persistently positive trend whereas with the same groups of people age has a negative relationship to learning gain (-10), to intelligence score (-24), and to initial performance score (-16).

Wanting to learn is the greatest aid to learning, interest in the subject to be learned aids the mental organization necessary for attention and retention. Motivation or zeal to learn increases the effectiveness of effort and practice in every learning test. Attitude, interest, and motivation are better sustained as age advances than is the speed of activity and they tend to channelize and conserve effort in the direction of organized patterns of experience. In adult learning groups, whether formal classes or informal social assemblies, these selective factors are at work and generally speaking, the older learners are those whose attitudes, interests, and motivations are most effectively focussed on the specific learning which they have undertaken.

AGE AND INTERESTS

From the array of activities, pursuits, and occupations observed and available in his environment every person selects those most often present most urgently pressed upon him, or most congenial to his inclinations. Gradually life behavior forms a recognized pattern in these terms and a man is known by his characteristic acts, interests, and achievements. Both formulated and unrecognized goals give direction and habit gradually conserves energy within limits favorable to progress toward the chief life objectives. Yet the result is by no means formally crystallized. Within the life channel age gradually brings certain features into relief and wears away others. The psychosomatic organism alters with age within the mold of behavior which it has created for itself, and as this occurs the outer expression too must alter. Thus it does normally without the loss of essential continuity.

Type of Interest Change. The change with age appears in the waning of active in favor of passive pursuits, of social in favor of individual recreations, of variety of interests in favor of a narrowing selection, of changing environments and objects in favor of a comfortable, settled routine. Interest in phys-

ical skill and daring adventure decreases definitely with age, disinclination to change of all kinds increases. Liking for linguistic activities decreases, but enjoyment of reading increases. Liking for people with desirable traits increases as does dislike for people with undesirable traits. The things liked best at age twenty-five tend to be liked better as age advances. The things liked least are liked less as one grows older. In each of eight occupations studied by Strong, who has reported these results, the same age trends have been found in parallel.¹⁷ Engineers, lawyers, life insurance salesmen, ministers, physicians and surgeons, school men, writers, and YMCA secretaries all "age" in interests along a slightly waning curve in terms of liking for activity and social extroversion, while preference slowly increases for quiet, comfortable, cultural pursuits. Rating in terms of conformity to their own occupational pattern changes very little. The differences persist between the eight occupational patterns and far outweigh age differences and age changes common to all.

Merging of Sex Differences. Individual, social, and cultural attitudes and interests differentiating the sexes show little age change for women in adulthood, while a marked trend inclines the men's decade scores gradually toward the women's mean. This tendency, noted by Terman and Miles, although persistent from early to late age, never obscures the sex difference.¹⁸ Even at eighty the attitudes and interests of men and women, although nearer together than at any other time since early childhood, are yet significantly and characteristically diverse. With increasing age the hobbies of men according to Landis change more than do those of women, which may contribute to the generally poorer adjustment of older men.¹⁹

AGE AND PERSONALITY TRAITS

The traits of youth are popularly known to be vigor, enthusiasm, self confidence, fearlessness, those of age are caution, frugality, devotion and persistence. Thoughtful observers know that stereotypes like these, while broadly meaningful, cannot be universally true or individually applicable. At every age individual personality differences outweigh age tendencies. As in vocational and occupational studies, so in personality appraisals by psychologic test methods, the evidence of persisting patterns throughout adult life is far greater and more significant than the evidence of age alteration in normal adults. When differentiated traits called neuroticism, self sufficiency, introversion, and dominance have been measured, in adults from twenty to eighty, constancy in the decade to decade means is the rule rather than the exception for both sexes.

Waning of Dominant Traits. A small trend, statistically significant for "dominance"—the active, social trait of determining rather than being de-

scales. The negative elements of the trait are social shyness, diffidence, willingness to be passive and let others take the lead. It is perhaps natural that men's development in our culture shows this slight waning of extraversion with advancing age. To the extent that it reflects a comfortable relaxing of tension in social adjustment, it indicates a normal and favorable change. If occupa-

tional and economic discouragement or a slackening of energy on retirement conditions it, then better health conditions, increasing social security, and more adequate planning for retired men may in time effect its elimination

Emotional Reactions at Critical Periods. Individual psychologic and psychiatric histories of older people may illustrate supernormal, average, and inferior developments Psychoanalyses of older adults show the possibilities and limitations in employing this technic in advancing age Studies of behavior at the critical periods of retirement or of the menopause indicate specific emotional hazards But there is need for new tests or new norms on present tests for adults of different ages before firm conclusions may be drawn^{20 21 22} Any remotely complete picture of the emotional traits of the aging personality as a whole derived from adequate numbers of studies of normal people is still waiting

OCCUPATIONAL AND PROFESSIONAL AGE RELATIONS

Whether men and women work because they must or because they will is a question no one can answer now It is certain that they are generally happier when they are employed and that with normal health they usually seek to remain employed to an age approximating three score and ten years The characteristics of youth are probably more favorable in certain occupations and less in others Forestry and fishing, extraction of minerals, manufacturing and mechanical occupations, transportation, communications and trade combined show, according to U S Census reports, a relative drop from the fifties to the sixties of 16 per cent The professions suffer a 4 per cent loss clerical occupations a 6 per cent gain, while agriculture, public services, and domestic and personal employment are increased almost 20 per cent

Physical energy, activity, and manual dexterity are variously involved in the professions as well as in the other occupations and age balance is maintained on the basis of these relative involvements Music, acting, and the show business are favorable for younger people Technical engineering, draftsman ship, architecture, and designing show early loss of numbers with increasing age Art, photography, and dentistry also decrease in numbers in the middle decades The learned professions show remarkable persistence of numbers up to the usual retiring age of sixty five and even beyond it

The recent war brought about a great increase in the employment of older persons Industrial management gave some attention to the selective placement of these workers because their labor capacity was seriously needed For sound psychologic and sociologic reasons this program of selective placement for nonhandicapped and handicapped based on job analysis and on examinations, and of check up appraisals of individual older employees, is essential^{23 24} Men are naturally hesitant about subjecting themselves to tests that may show occupational or skill decrement with age and so perhaps threaten their economic security Studies that have been made show trends similar to those already indicated in the measurement of the basic functions, skills, and intelligence A few results are available comparing work output of younger with older workers in a variety of occupations These generally show speed or output decrement with increasing age But if the older worker's placement has been in terms of favorable mechanical and environmental factors his productivity may continue at a high level

Role of Experience. In the age of craftsmanship, experience counted for more than speed. In a machine age, young men may feel that they are more effective than older workers because they are faster and lose fewer work days. It is in the directive and administrative positions that the experience and judgment of older men come fully into account. Where older industrial workers are shown to have a higher output than younger men at the same jobs, selective factors account for the difference. Only the best older workers have been retained. Perhaps equally good men have been drawn off into higher positions, certainly poorer ones have failed to keep their places. Under systems designed for complete utilization of manpower the goal is the utilization of workers of every age in a selective service that employs their optimal skills and energies without unfavorable competitive elimination. Under such a system studies of the relation of age to production could be meaningfully made and utilized.

Labor turnover is expensive to individuals and to industry. Older men are steadier in their jobs, require less frequent replacement, and are therefore less expensive in training. They have fewer industrial accidents relative to hours worked and are more careful with equipment and less wasteful of materials. Older workers are less distracted by social interests and they tend to develop a strong sense of loyalty and responsibility. Sick leave in industry correlates with the greater incidence of morbidity as age advances. Duration of illness shows a greater correlation with age than does illness incidence. Sickness may influence work attitude unfavorably in the direction of discouragement. Time out decreases experience and practice and so lessens the characteristic assets of older workers. Recovery from accidents is less satisfactory at older ages, but older workers, although more liable to disease, show their greater caution in a smaller accident rate.

In spite of the age losses no one of the professions, and indeed no one of the occupations, as reported by the census figures, excludes workers at sixty, seventy, or any reported age. As these figures represent the individual's own statement of his occupational or professional classification, they seem to give evidence of the *will to work* as well as of the *need to work* among older as well as younger men. In the old there comes eventually the need for retirement. Preparatory planning for this phase of life can and should be so realistic as to avoid any experience of traumatic shock in separation from office or factory.^{25 26 27}

AGE AND GREAT ACHIEVEMENT

"Men are at their best when enthusiasm and experience are most evenly balanced." The world's tasks require not only the best effort of workers at peak achievement age, however, but also the vigorous contribution of young learners and the deliberative and conserving influence and example of older men. In no field of labor is the "opus magnum" accomplished by men in a single favored decade. The curve of rise and decline for master work in science, art, literature, philosophy, and statesmanship follows the characteristic pat-

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ment of younger men. Record holders are most often in their twenties, but in certain skills of sport, as in marksmanship, achievement continues throughout middle age. Curves or statistics indicating the optimal semidecade for masterpieces of poetry (late twenties), science (early thirties), art, music, mathematics and prose writings (late thirties), astronomy (early forties), show how spontaneity, originality, and youthful energy are balanced against breadth of knowledge, experienced judgment, and persistent continuity of interest in these several fields of human endeavor. Public leadership in defense, education, government, and religion tends to reach its peak somewhat later than individual creative production or invention. Great military and naval commanders have most often been distinguished in the late forties and early fifties. American college presidents serve most frequently in the early fifties, cabinet members in the early or late fifties, commercial and industrial leaders and presidents of republics in the late fifties, and presidents of religious groups in the sixties.

The peak values are interesting and significant, but they do not tell the whole story. In every field of physical activity and intellectual endeavor young men and also old men are at work. Masterpieces have sometimes come from the precociously mature and they have not uncommonly been the work of men of great age. Allowing for population differences, more than 25 per cent of great works and important inventions have been the accomplishment of men over fifty. And the sixties and seventies have each given their share. In administrative service, a quarter or more of the greatest achievement may occur after age sixty. In work below the "unique" or "opus magnum" level, including much of the total output of great men, as well as the bulk of human achievement, production continues through the life span to sixty-five or seventy years, in most instances where health is normal. The pertinent conclusion, for average as well as for specially gifted people, is that good work is not limited by life age, although in specific amount and in distinguished quality a measured decline occurs, especially in certain fields, with advancing years. Ambition, enthusiasm, and alert energy contribute to the accomplishment of youth. Continuity of experience, persistence in effort, and a developing sense of responsibility give their effective stamp to the work of age. Individual variations occur at every age and in every field of action and thought. Life achievement is not just a matter of peak production. Training and servicing are highly essential. Routine work can be done effectively at every age and the old have their place in this as well as in the directing and organizing of the work of others, in which some of them are especially competent.

CONCLUSION

Society, whether industrial or social, functions most adequately when all ages are represented in its structure. Young workers are stimulated by competition, but they need also the sustaining assurance of relative permanence in service. Old employees give stability and continuity to the life of industry and in turn derive psychologic encouragement and effective invigoration from contact with younger associates. Similarly, social life is enriched by the presence and interrelationships of age series. Child, young adult, middle aged adult or parent, and old adult or grandparent, each gives to the others the sense of the continuing life values that wax and wane in varying degrees from

birth to the end of life. The goal of individual and group hygiene is the adequate function of each individual in the community. For efficient industrial organization toward its objective, optimal production, the availability of every unit of manpower is essential, and for social progress the highest level of development and welfare in the function of the individual and of the social community is the final aim. Every life age has its part to play and its distinctive contribution to make.

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CHAPTER 6

MEDICAL CARE OF NORMAL SENESCENTS

EDWARD J. STIEGLITZ

GERIATRIC medicine deals with the preservation of health of the aging and the aged as well as with overt disease. Aging may be normal or abnormal but there can be no sharp line of distinction. Apparently normal aging brings about many changes in structure, functional capacities, and in the mind. Most of these gradual and insidious changes have been discussed previously in the preceding chapters. Physicians may be aware of the significance of these alterations in physiologic economy and of the need for gradual modification of hygiene and the manner of living, but lay patients are not so informed. It is admitted by everyone that it is wise and sensible for the child to prepare to become an adult, but almost no young or mature adults make the slightest effort at preparation for senescence and senility. In far too many instances some serious breakdown forces people to pay attention to the limitations of their age. Many such breakdowns are unnecessary if foresight be but applied. It is one of the most important functions of geriatric medicine to supply wise guidance and instruction in adaptation to advancing age.

Health, like age, is relative. There are degrees of health just as there are degrees of illness, or of intelligence, or equanimity, or beauty. Health is an abstraction or ideal. Perfect ideal health is probably unattainable, though it may be approached. Unfortunately, the antiquated, negativistic definition of health as "that state of being existing in the absence of disease" is still to be found in some medical dictionaries. Let us redefine health as having quantitative attributes and perfect health as that state in which all the functional capacities of the organism have maximum reserves. Optimum health is affected by age. The adolescent or young adult may have maximum cardiac and muscular vigor, but the intellect has not as yet developed to its peak. By the time intelligence and emotional homeostasis are fully developed, somatic depreciations will have reduced other functional capacities to below optimum levels.

The relativity of health is particularly significant in the practice of geriatric medicine, for it closely parallels the variable relativity of biologic age. Normal is a vague and misleading term. What does it mean? It means different things to different people, but the most common connotation is that "normal" is nearly synonymous with "average." Average certainly does not imply optimum. Thus, normal health and optimum health are frequently widely divergent.

From the beginning, modern medical education has been concerned chiefly with teaching oncoming generations of physicians to identify and treat disease entities. It is only recently that attention has been focused upon the individual who may or may not be suffering from an indentifiable disease. Pediatrics and psychiatry are the two fields of medical practice in which concern with the patient rather than with his disease has met with the least re-

sistance and inertia. The prophylactic value of mental hygiene is becoming widely recognized. Today there are innumerable "Well Baby Clinics" throughout the land; pediatricians have guided many thousands of apparently well babies to greater health. But where are there available similar services for adults and especially for senescents? A few health centers approximate the necessities of such guidance, but far too few. The situation can be improved, even if an ideal optimum is never fully approached. The critical age in geriatric medicine is the two decades from forty to sixty years.

CONSTRUCTIVE MEDICINE

Treatment of disease is essentially effort toward the *reconstruction* of destroyed health. The *construction* of greater health in those relatively well may be appropriately called constructive medicine.¹ Constructive medicine is more than preventive medicine, though it includes all of the activities of the latter.² Preventive medicine, by its very definition, is concerned with the prevention of disease; mere avoidance of disease does not add to health.³

Constructive medicine, like preventive medicine, may be applied by either what we may call wholesale or public measures, or by a retail or personal approach. Sanitation, quarantine, and the like have proven of immense importance in the control and near eradication of the communicable diseases of childhood and youth. But conventional public health methods cannot suffice in a preventive or constructive attack upon the degenerative diseases so significant in later maturity.² We have emphasized repeatedly the importance of the factor of individual variation. No amount of control of the external environment can prevent arteriosclerosis, diabetes mellitus, many forms of malignant tumors, hypertension, or gout. These are endogenous disorders. Their prevention is an *individual* problem, demanding initiative and effort on the part of the individual benefited.

One of the major reasons why the environment control methods of public health and sanitary engineering⁴ have been so dramatically successful in reducing morbidity from communicable diseases is that almost no individual effort was required on the part of the beneficiaries. Today, clean water and food are taken for granted—often too much so, obesity due to excessive food consumption is an etiologic factor in the vascular, arthritic, and metabolic depreciations of later years. Education in wiser living habits, including guidance in nutrition, is the one "wholesale" technic which has promise of considerable accomplishment in prevention of the degenerative disorders.² The periodic health inventory and consultation is the avenue through which *personal, individualized guidance* can be applied. As such instruction is *sought* by the patient, education of individuals in matters of health is one of the most important technics of constructive medicine for adults. And constructive medicine is the highest form of preventive geriatrics.⁵

PERIODIC HEALTH INVENTORY

The periodic health inventory is the keystone of the arch of geriatric

ciency and treat them promptly, and (3) to teach the patient how to use his endowment of health more wisely

The choice of the term "inventory" instead of "examination" is deliberate. For a properly conducted inventory includes much more than a clinical physical examination. All too frequently periodic examinations are superficial, hasty, and carried out carelessly because of lack of interest on the part of the physician. It is small wonder that in many quarters the concept of periodic examination has fallen into some disrepute. The findings of a physical examination, recorded and filed away upon a card, do the patient little good. A detailed, painstaking history, functional tests of reserve capacities and laboratory studies are requisite to proper thoroughness. If health inventories are so conducted and sound, carefully considered advice is given the patients, there should be no reason for lack of patient confidence. However, it must not be forgotten that mensuration of health requires higher diagnostic acumen and more detailed data than are necessary for the diagnosis of frank disease. The average layman expects an examination to be complete in a half hour, but any one concerned with business knows that a thorough "inventory" is far more time-consuming. Cooperation is greatly improved by the simple substitution of phrases. But it remains the responsibility of the physician to see that his studies are thorough and his guidance and educational efforts sound. In no other manner can constructive geriatrics advance.

Comprehensive health auditing is far more difficult, time consuming and expensive than the diagnosis of disease. This must be appreciated by physician and patient alike if the full potentialities of constructive or preventive geriatrics are to be realized. Intimate and prolonged cooperation is essential. The initiative for such constructive consultations must come from the patient. More and more patients are demanding this type of service. Many complain that the majority of physicians they have consulted have scoffed at these concepts of prevention and health construction. Intellectual inertia of clinicians has retarded the development of the potentialities of this truly significant clinical instrument. To illustrate by a particular instance:

A former patient of the author moved to Boston where she continued her habit of seeking an annual inventory but was told by one of the more famous of the clinical professors that if she had no specific complaints such an examination was unnecessary. However, he performed a perfunctory physical examination, discovered an apical systolic murmur, and told the patient she had serious rheumatic heart disease and had better be careful. The lady, some forty-two years old, was frightened. She immediately made an extended trip to see her former physician, who likewise heard the apical systolic murmur but was sufficiently experienced in health mensuration to include a blood count in her examination and discovered her hemoglobin to be but 62 per cent of normal! The first physician had not bothered with such a trifle. Correction of her anemia (due to preclimacteric menorrhagia) promptly caused the complete disappearance of the innocent hemic murmur.

It is instances such as these which reflect adversely upon the medical profession. There is no excuse for careless, superficial study of any patient.

Limitations and Potentialities. Obviously, periodic health inventories have their limitations as well as potentialities. Perhaps the most serious of the factors which limit the application of the procedure is that of cost. A proper inventory is and must continue to be, fairly expensive because it is a time-consuming study requiring rather exceptional skill. Attempts to short cut or prune various items from the inventory, with the idea of effecting economy of

admitted 'to see how your retinal hemorrhage of six weeks ago is getting on. He had not mentioned this previously for fear of frightening his friend.' The patient's convalescence was delayed by the discovery of a mild diabetes, controllable without insulin. His return to practice has been only partial and he now complains much of failing memory.

Had stress tests, such as the glucose tolerance test and effort response been carried out at the time of his original inventory, his potential diabetes and arteriosclerotic heart disease would have been discovered.

Functional studies need not be elaborate. Simplicity, safety and most of all, sound logic in creating true conditions of physiologic stress are the primary criteria of tests for functional reserve capacity. Details of various methods must, of necessity, be omitted here, but it cannot be mentioned too often that *in order to detect early depreciations which are as yet amenable to therapeutic correction, they must be searched for.*

Because early functional impairments, brought on either by disease or senescence *per se*, are largely asymptomatic it is important to follow up all minor clues in diagnosis. The silent and insidious onset of most of the so-called degenerative diseases often causes serious delay in older patients seeking medical advice. Only disorders which hurt receive prompt attention. Frequently pride, fear, or wishful thinking causes the elderly individual wilfully to conceal symptoms. Particularly is this true of aging men who refuse to recognize and/or admit the depreciations of senescence. The sin of deceit is rarely one of commission, however. On the other hand, wilful omission of symptoms is very common among elderly patients. It is most unwise to rely solely upon volunteered information. The patient's history must include a searching inventory. This is frequently best obtained by analyzing functional adequacy of the major systems by specific leading questions. Such systemic inventory often yields information of the greatest moment which would not be elicited by the patient's volunteered complaints. For example, the patient may complain of increasing dyspnea, apokamnosis, and languor, but fail to mention bleeding upon defecation. Yet bleeding hemorrhoids (with the invariable potentiality of rectal cancer) may be responsible for the dyspnea by causing secondary anemia. A history of migraine is rarely volunteered. These patients have had their distressing attacks for years and their mothers before them, they don't expect or seek relief from their migraine. Yet our problem is the patient as a whole rather than palliation of prominent symptoms, and the coexistence of a migrainous constitution⁸ may materially alter diagnostic interpretations. The hypertensive or arteriosclerotic patient will rarely mention recurrent indigestion or the habit of taking sodium bicarbonate after meals, though the coexistence of peptic ulcer is not uncommon. The discovery of secondary lesions is predicted upon following up all such apparently minor symptomatic clues.

Organization of the Inventory. A well organized, systematic plan of study is essential if all the important sources of information are to be included. The basic, minimum outline can be modified to fit the individual patient by expanding any phase as indications for more extended study arise. Any static routine is to be avoided, such will be either too superficial to be thorough or so elaborately comprehensive as to be prohibitive in cost. Flexibility is maintained by working from a minimum designed to reveal potential difficulties. This minimum is discarded upon the slightest suspicion of deviation from the optimum.

The effectiveness of an inventory of health is measured by the wisdom of the guidance offered and the encouragement of motivation and instruction of the patient so that the advice may be followed intelligently. For wise guidance, our information must be comprehensive as well as accurate. For intelligent cooperation on the part of the patient, our instructions must be specific and meaningful. People will follow advice if they know *why* they are doing such and so. Suggestions are usually quickly forgotten if the reasons for making them are not explained.

The minimum procedure of the health inventory involves five major steps. These are subdivided and organized as follows:

- 1 History
 - 1 1 Present, clinical
 - 1 2 Past, clinical
 - 1 3 Past, occupational
geographic
familial and marital
 - 1 4 Present, systemic
 - 1 5 Habits, including anxieties
- 2 Physical examination
- 3 Laboratory and x ray studies
 - 3 1 Minimum laboratory
 - 3 2 Minimum x ray
 - 3 3 Electrocardiogram
 - 3 4 Basal metabolic rate
 - 3 5 Stress tests
- 4 Supplemental studies as may be indicated by preliminary observations
- 5 Guidance
 - 5 1 Report to patient on findings, both normal and abnormal (summary of evaluation in detail)
 - 5 2 Interpretation of significance of observations (analysis)
 - 5 3 Specific items amenable to therapy
 - 5 4 Hygiene
 - 5 5 Future outlook, analysis and modification of plans
 - 5 6 Questions by the patient

History. In many respects the patient's life history is the most important source of information regarding the present level of health, biologic age, and outlook.⁹ *We are today what we are largely because of what happened to us in our yesterdays.* The older we become the more significant are these yesterdays, there are more of them.² Therefore, all parts of the history should be elicited with conscientious attention to detail and sequence. Older people are often garrulous and somewhat confused regarding dates and sequence of events, but with time and patience the grain can be separated from the chaff and extremely valuable information elicited. It is important that the patient have full opportunity to tell his or her story, there is probably more dissatisfaction on the part of the patients with the application of the health inventory because of a hurried history than because of any omissions in the physical examination. If the inventory is to be effective at all, the patient must feel confident that the doctor fully appreciates all the details of his problems.

Doubt, in contrast to conviction, means that instructions will not be carried out and therefore therapeutic results become unsatisfactory.

In connection with *present subjective complaints* we are particularly interested in the story of their onsets. It is at the beginning of a disorder that we are most liable to find etiologic correlations. The patient's own opinion as to etiologic factors is worth listening to, his conclusions may be wrong but in presenting the reasons for his opinion most useful information may come to light. Insistence on knowing something of date of onset, frequency of recurrences, cyclic phenomena, correlations with physiologic activities, and sequence of events may be time consuming, but it is necessary if we are to avoid gross errors in evaluation.

The patient's medical *past* is important. A detailed listing of previous illnesses should include information as to sequence (age of episode), intensity, duration of each illness, tendency to recurrence, and, if possible, something regarding the therapy applied. It is at this point that the patient frequently reveals for the first time an intolerance to certain medications, poor reactions to anesthetics or to the sulfa compounds are facts worth noting. We should be particularly interested in those disorders most likely to leave sequelae. Specific inquiry regarding rheumatic fever and influenza in the epidemic of 1918-20, repeated tonsillitis, and allergies should not be omitted. Unless these are asked about specifically, the past history is very likely to be incomplete, people forget readily those unpleasant episodes which they don't want to remember. The role of epidemic influenza as an etiologic factor in the causation of vascular disease has not been appreciated by many. In the years 1920 to 1935, a considerable percentage of mature patients with hypertensive disease, chronic nephritis, or myocardial damage volunteered the statement that "prior to the flu I felt well, since then I've gone downhill." That a similar, though less severe, postinfluenzal damage may be etiologically related to some of the degenerative disorders seen today is quite probable. Virus infections characteristically have a long drawn-out period of convalescence; the "hang-over" is typically much worse than the apparent severity of the acute attack appeared to warrant. From the viewpoint of late and silent parenchymatous injuries the virus infections are significant.

The past history should include also a brief review of occupational history, education, and marital background. Occupational and educational backgrounds are important factors in planning for wise investment of the increasing leisure which is so often a problem in later years. A series of divorces, or a long happy married life recently rudely shaken by the death of the mate, is definitely pertinent to present emotional problems. Knowledge as to where the patient has lived (geographic history) may be most useful in connection with adaptation and exposure to specific hazards (rheumatic infections in the North, certain parasitic infections in the South).

The third part of the history, an inventory of symptoms by systems, is frequently most illuminating. By specific inquiry as to functional effectiveness and response to physiologic effort, we are most likely to discover early depreciations in reserve capacities. Secondly, items otherwise forgotten are brought to light. Frequently patients have experienced some abnormality for a long time, have repeatedly failed to obtain relief, and thus expect to suffer all the rest of their lives, migraine headaches, chronic leukorrhea, constipation, nocturnal urinary frequency, and habitual apokamnosis are among the

items most frequently unintentionally omitted from the volunteered complaints and elicited by direct inquiry

The systemic inventory should be sufficiently inclusive to indicate whether the various organ systems are functioning normally. Many different outlines have been constructed. The following schedule for minimum routine inquiry has been found to be effective, though other arrangements may do just as well.

SYSTEMIC INVENTORY

C N S	Sleep	Rested	
	Headache	Location	
	Frequency	Time	
	Associated with		
	Syncope	Paresthesia	
	Vertigo	Ears	
	Vision	Lenses checked	
Respiratory	Coryza, frequency	Scotomata	
	Pharyngitis	Epistaxis	
	Cough	P N drip	
		Night sweats	
Circulatory	Undue dyspnea		
	Card consciousness		
	Edema		
	Reaction to environ temp		
G I	Appetite	Weight	
	Distress p c		
	BM x	Catharsis	Stools
	Nausea	Icterus	
G U	Frequency n	d	V D
	Urgency	Dysuria	
	Mental		
	Menses, onset at	Last period	
	Interval	Amt	
	Pain	Premens Intox	
	Leukorrhea	Preg	

It should be noted that many of the specific inquiries are directed toward eliciting data as to responses to stress. Tolerances to extremes of environmental temperature, and the extent of exertion necessary to induce dyspnea, reveal reserve capacities. Dyspnea on effort is not abnormal or indicative of lowered reserve if the effort necessary to induce hyperpnea is great enough. In the day by day experiences of the patient, we have what is probably the best cardiac function test yet devised. (See Chapter 24.) To be sure, undue dyspnea will usually be rationalized by being attributed to smoking, sedentary habits, or obesity, but the fact of lowered circulatory reserve remains. The true explanation must be determined by the physician, the phenomenon is a challenge to his diagnostic acumen. Anemia is as likely to be a factor as tobacco.

The sleep pattern may be most suggestive of emotional difficulties and of the role of psychogenesis in actions, and the like, aid in evaluating the status of older adults. (See Chapter 2.) Health is profoundly affected by living habits. The following outline is a useful minimum.

HABITS
Fluids
Milk
Coffee
Alcohol
Tobacco
Drugs
Exercise

Spices
Salt
Meat
Work
Worry
Leisure

The habitual consumption of beverage fluids is too frequently ignored in queries concerning dietary customs. Older people, whose renal ability to concentrate the urine may be impaired, very frequently consume far too little water (see Chapter 37). By this question alone the full explanation of chronic constipation may be elicited. The suspicion of calcium deficiency is aroused when habitual milk consumption is negligible. Habits of work and play, relaxation, boredom, use of leisure, and exercise are all significant. Frequently, discussion of these items brings forth the most essential part of the patient's story—discontent with occupation, with home, and/or with self. In such discontent may lie the whole explanation for such situations as obesity, anorexia, or hypertensive disease.

The family history may reveal useful information, but too much reliance must not be placed upon it. Negative evidence is not proof of absence. We must remember that the knowledge of patients concerning the diseases and causes of death of other members of the family is very limited and, more often than not, inaccurate. Furthermore, at best we cannot expect to go further back than the previous generation. No student of eugenics would attempt to draw any conclusions anent hereditary characteristics with data from such a short segment of the family tree. It has been my experience that no more than 10 per cent of individuals know the cause of death of their four grandparents, and I suspect that of these 10 per cent at least half the answers are wrong. Humanity is of such mixed ancestry that only when a disorder appears several times in a family is it truly significant in indicating heightened vulnerability of the individual patient.

However, every source of possible information must be explored and the family history should not be neglected. Interest should not be limited to the causes of death of parents, grandparents, and siblings. Migraine and gout are not fatal disorders, but their hereditary transmission is well known. Of particular importance are the habits acquired in childhood from the family environment.¹⁰ Obesity may or may not be biologically hereditary, but it is frequently familial because of the child's exposure to the unwise eating habits of his parents.⁶ Habits persist longer than life; they are transmissible from generation to generation. Similarly, anxiety is contagious and a childhood environment in an unstable home filled with apprehension is the forerunner of many adult neuroses.

In brief recapitulation it should be emphasized that in geriatric medicine more than in any other field *multiplicity of etiologic factors* is the rule. Each and every possible source of information must be explored and exhausted, for the greater the knowledge concerning etiology, the sounder is prognostication, and the more logical and effective is therapy. In later maturity and thereafter complex superimposition of several disturbances, often preexistent and occult, is the rule rather than the exception. Age vastly complicates the intricacy and multiplicity of etiologic factors to be considered in diagnosis.

The reciprocal effects of several functional depreciations often alter the manifestations of disease in the aged

Physical Examination There is little need to discuss here what constitutes a thorough physical examination. However, it should be stressed that the examining physician must be acutely alert for subtle hints in examining a patient for the purpose of health evaluation. Obvious and conspicuous signs of illness are not to be expected. Minor evidences of disordered physiologic activity and functioning are particularly significant. Acuity of perception on inspection is essential.

For example, the texture of the hair, its oiliness or dryness, may be the first clue to discovery of a relatively minor variation from normal in thyroid activity, and if combined with dryness and a leathery texture of the skin justifies confirmation by determination of the basal metabolic rate. The basal metabolic rate is the only physiologic constant known to change notably with aging and for which we make correction for age in our calculations of normal. After forty, the so called normal decline in oxygen consumption is frequently exaggerated. Low basal metabolic rates for age are observed in a considerable percentage of senescent patients free of obvious evidences of hypothyroidism. Correction of such deficiencies may mean the difference between habitual fatigue and more nearly optimum vigor and endurance. Hypothyroidism may be a factor in making a hypochromic deficiency anemia resistant to ordinary nutritional therapy.

Inspection of the ocular fundi should never be omitted from the physical examination. The status of the retinal arteries is most important in evaluating the extent of arteriosclerosis. Here we have a magnificent opportunity for direct inspection of smaller vessels whose slow, asymptomatic deterioration is of immense importance to the senescent individual. Every feasible source of information regarding the functional and anatomic status of the cardiovascular system should be explored.

It does not suffice to simply listen to the heart at rest. A simple exercise response procedure should be carried out in all instances. Elaborate apparatus is unnecessary, but keen observation is requisite. A useful procedure is to note the pulse and blood pressure at rest and then again immediately after such a simple exercise as bending down to touch the floor several times. Any reaction showing an excessive acceleration of the pulse rate or a drop in the systolic pressure is indicative of lowered cardiac reserve. Such an observation does not demonstrate the anatomic nature of the cardiac disorder, which may be coronary arterial disease, myocardial flabbiness, or a valvular lesion, but it does make further study of the heart obligatory.

Relatively few physicians measure the blood pressure on both arms. Asymmetry of more than 5 to 10 mm Hg is common enough to warrant bilateral determination as a routine procedure at least once on every patient.¹¹ Decided asymmetry suggests some aortic distortion, either congenital or due to disease, or perhaps a cervical rib. A false conclusion of asymmetry in the arterial tension may arise unless the pressure in both arms is determined.

the standards for optimum weight presented in Chapter 2. In a health inventory we are concerned with optimum health rather than a discovery of gross evidences of disease. As age advances, obesity becomes an increasingly serious menace. Weight reduction is much more readily accomplished when 5 to 20 pounds should be lost in the forties than when more should be lost in later life. The time for the obese to start on a weight reduction program is today, not tomorrow.⁶

These are but a few illustrative examples of the type of information we seek in a health inventory. It should be quite obvious that pelvic and rectal examinations should be routine; the recent emphasis on cancer control by earlier discovery should remind every physician to investigate thoroughly any unusual vaginal discharge. Keen observation during the course of an examination frequently reveals much indirectly: unusual hyperpnea following the effort of climbing onto the examining table; vasomotor instability as indicated by flushing and/or hyperhidrosis; fine tremor; slightly viridescent pubic hairs; and the like are all significantly suggestive.

Laboratory and Supplemental Examination. Clinical laboratory studies are a necessary and often illuminating part of the health inventory. However, just because methods and instruments of precision are employed, we must not give these data unwarranted prominence. The judgment applied in evaluating the observations is vastly more important than the findings themselves. A tendency to let clinical laboratories make diagnoses on the basis of their observations is evidence that the clinician is either unwilling or incompetent to do his own thinking. The data must fit with the clinical picture. Frequently, combinations of observations are much more revealing than isolated findings.

The minimum laboratory and supplemental studies compatible with comprehensiveness sufficient to truly evaluate the health status of mature patients include the following:

- | | |
|--------------------------|---------------------|
| 1 Blood | 2 Urine |
| Hemoglobin | Reaction |
| Red blood count | Specific gravity |
| White blood count | Albumin |
| Sedimentation rate | Glucose |
| Complement fixation | Sediment |
| 3 Basal Metabolic Rate | 4 Electrocardiogram |
| 5 Stress Tests | 6 X ray |
| Renal concentration test | Chest fluoroscopy |
| Glucose tolerance test | Dental films |
- Frequently desirable though not routinely requisite are:
- Blood cholesterol concentration
 - Blood uric acid concentration
 - Mantoux or tuberculin patch test

Other procedures such as gastrointestinal x ray study may be indicated by the history or findings on physical examination.

A differential count of the blood is probably not necessary as a minimum requirement though it should be done whenever there are abnormalities in any of the routinely included observations. The hemoglobin and sedimentation rate determinations should never be omitted under any circumstances. Mild degrees of anemia are very common. They are significant for the objec-

tive of the inventory is to evaluate health and anything less than optimum is worthy of attention. We must not be satisfied with average or so-called "normal," the majority of adults in their later years are below optimum but with proper management can attain better health. Any hemoglobin concentration below 90 per cent of optimum deserves therapeutic attention.

To many, a routine basal metabolic rate determination may appear unnecessary. However, it has been our experience that a great many patients aged over forty reveal rates 15 to 20 per cent below normal without obvious signs of myxedema and that they profit greatly from the judicious administration of thyroid substance. Thyroid substance should never be prescribed without accurate measurement of the basal metabolic rate and recheck one month later to make certain that the dosage is precisely appropriate to the patient. No two people respond to thyroid substance precisely alike. It has proven helpful to explain to the patient that, just as in navigation, it is wise for the pilot or navigator to determine his position from time to time. There are so many places in clinical medicine where we must rely on estimation and judgment alone that I feel grateful for the opportunity to measure accurately.

The electrocardiograph, like any other instrument, can give very misleading results. It has been shown (see Chapters 24 and 25) that severe cardiac damage may exist in patients yielding normal electrocardiographic tracings and also that gross abnormalities can be present in the tracings without there being any other evidences of cardiac disease. Therefore, we must be cautious in not reading too much into minor variations from the accepted normal. The interpretation must fit with the clinical picture as revealed by the history, and physical and fluoroscopic examinations.

The renal concentration test (see p. 591) as a formal procedure is often unnecessary if single specimen urine reveals a specific gravity of 1.025 or more. As the preparation for the basal determination involves omitting breakfast, it simplifies matters to conduct the renal concentration test on the same occasion. Blood analyses for NPN, urea, etc., are not indicated unless definite evidence of renal incompetence is elicited, increased concentrations in the blood are a sign of renal failure and will *not* appear early. The chief value of the concentration test is that it measures the response to physiologic stress and is thus an index of functional reserve.

X-ray examination of the chest should be included in every health inventory. Tuberculosis is wrongfully considered a disease of the young, many students of the disease feel that chronic, and largely asymptomatic, pulmonary tuberculosis in the elderly constitutes one of the gravest sources of contagion today. Furthermore, it is very useful to have a 2 meter chest film as a permanent record of cardiac diameters for comparisons with future findings.¹⁴ Contrasts over the years are most significant when compared to an early baseline. In many respects, fluoroscopic examination of the chest is more desirable than obtaining only an anteroposterior film. By using preliminary fluoroscopic examination we may omit roentgenography for those patients with perfectly normal cardiac contours and measurements and with normal pulmonary fields, and thus reduce the cost of x-ray examination. Furthermore, rotation of patient in front of the fluoroscope gives one the opportunity to study the arch of the aorta and the mediastinum. An anteroposterior film of the chest occasionally fails to reveal a saccular aortic aneurysm. Mobility of the diaphragmatic leaves is also worthy of note.

Guidance. The primary purpose of any diagnostic study is to assist therapy. Interpretation of findings, advice toward more nearly optimum mental and physical health, and assistance in maintaining vigor are what the patient seeks. The value of the whole procedure is determined by the wisdom, clarity, feasibility, and foresight of the advice offered. All the rest of the inventory is but the foundation, and as such is useless if the suggestions are not such as can and will be followed.

This phase of the periodic health consultation is conveniently divided into five parts: (1) Evaluation of findings, (2) Correction of specific defects in health, (3) Hygiene of living, (4) Discussion of future plans, and (5) Questions.

Interpretation of Observations. The patient is entitled to know and needs to learn what was discovered by the diagnostic studies. A quick summary of essential observations, normal as well as abnormal, is useful if only to increase conviction that there are adequate facts available for the conclusions. Only intelligent adults will seek such periodic inventories; their intelligence should not be insulted by superficial discussion. For example, it is valuably reassuring to the patient to learn that because of the high specific gravity of her urine, normal blood pressure, and satisfactory hemoglobin, we can be certain that the eclampsia which she had gone through ten or fifteen years previously left no significant renal damage.

Mere recapitulation of the factual observations does not suffice. We certainly cannot expect a patient to evaluate the importance of this or that deviation from the assumed normal, and, unless a wise appraisal is made, a periodic health inventory has the potentiality of doing much harm by inducing hypochondriac fears over insignificant changes. Of particular importance in this most difficult part of the audit is a quantitative attitude toward the changes observed. It does not suffice merely to name an abnormality discovered, whether it be a subtle depreciation in reserve capacities or a clinically definable disease entity. Diagnosis of health, as of disease, must include impressions regarding causation and degree of change as well as character of the change.

Interpretation of the personal significance of findings must be comprehensive and expressed in such manner as to be clear to the patient untrained in the biological sciences. Here the use of similes is of great value; the motor car is a useful object to illustrate disorders of metabolism or the circulation. Hyperthyroid activity can be likened unto a throttle too widely open, lowered metabolism to too much "choke" in the carburetor.

A frequent finding which demands interpretation is *obesity*. The patient needs to be informed of the increasingly detrimental influence of obesity as age advances, of the statistical evidence that obesity adversely affects longevity, of the mechanisms involved in its causation (frequently psychogenic excessive food consumption), and of the ill effects upon the cardiovascular apparatus. We should not expect the patient to make the sustained effort necessary for wise weight reduction unless he understands the *reasons* for so doing. The more clearly the patient comprehends *why* he is to do so and so, the more intelligently can he apply the advice proffered. Similarly, the relative insignificance of minor variations in the arterial tension should be explained to allay the apprehension so commonly *created* by physicians. A frank discussion of what is meant by a lowered cardiac reserve, in contrast to the

and absolute specter of "heart disease," will prevent the formation of a cardiac neurosis, and at the same time lead to wiser use of the impaired structure

Correction of Specific Defects in Health Anemia, dental infection, hemorrhoids, constipation, hernia, varicosities, obesity, hypothyroidism, and the like, are amenable to direct therapy and correction. These should be attended to, both because of the local disorders, and because of consequences upon the individual as a whole. Hemorrhoids may not be causing much pain but it is rather pointless to try to correct a secondary anemia in the presence of daily bleeding. The management of constipation is usually largely a matter of education in habits of eating and drinking (see Chapters 34 and 35). The patient rarely realizes that the character of what comes out depends more upon the character of what goes in than upon any abnormality of the bowel itself.

The control of the chronic progressive disorders which may be discovered in their incipency by the health inventory should be outlined. The patient

benefit for many years. The diabetic, likewise, needs education as much or more than a rigid diet order and a prescription for insulin. These are merely illustrative of the therapeutic problems that will evolve from adequate health inventories.

Hygiene of Living The ignorance of most people as to how to use the machine which is their body, and which houses their mind, is truly appalling. As Artemus Ward has said, "Ignorance is not not knowing, but knowing so many things which ain't so." Education in proper use of the physical organism and discipline of the psyche is perhaps the most important part of the health consultation. Here we have an opportunity for direct, personal, individualized teaching, or virtual tutoring. Often it is possible to arouse curiosity so that the patient will seek further information in nontechnical health educational literature.^{6, 15} Advice on diet (see Chapter 12) should not neglect the importance of an adequate water intake. This is too often forgotten. Older people are very prone to exist on diets too low in protein and with insufficient fluids. Questions as to sleep, smoking, exercise, exposure to sunlight, habits of work and play all warrant attention.

A usually neglected, though very important, part of periodic health inventories is a careful, considerate, but searching study of the emotional balance of the aging patient. Senescence brings many complex emotional problems to the fore in such a manner that they rarely cause primary complaints but may be most significant sources of psychosomatic manifestations. Prophylactic psychiatry in later maturity is a potent weapon in diminishing the ill consequences of psychic stress. Occupational or employment conflicts are common, but frequently preventable, sources of emotional upset. The involuntional psychoses are more effectively treated if detected early. Evaluation of mental

laxis. The anxieties and indecisions of senescence are a distinctly important field of mental hygiene.⁶

The wise investment of leisure raises many significant questions which

vary greatly with age. With aging comes more leisure. Constructive, planned use of such leisure favorably affects the physical and mental health and happiness of senescent individuals. Two important groups of factors contribute to a great increase in leisure among the aging. These should be distinguished, for they raise different problems in guidance. Normal senescence brings about an inevitable slowing of the pace of life, the increase in leisure is then gradual. Illness may cause relatively abrupt cessation of former occupational activities, thus liberal leisure may be introduced so suddenly that spontaneous adjustment is more difficult. Every discerning physician has observed that there is more distress and/or unhappiness in the chronically ill due to boredom engendered by restriction of activity enforced by the physical disability than distress or pain from the disease *per se*. Boredom is a dangerous mental state. It is, also, wholly unnecessary.

In childhood and early youth leisure is employed to release surplus physical energy. After adolescence sports and active games do not suffice alone, more complex interests arise. The awakening of sex introduces music and literature in new lights, curiosity is often crystallized and ambition toward the future may greatly alter the spontaneous uses to which leisure is put. During the fruitful years of maturity the concerns of everyday life, the necessity of earning and mating, and the responsibilities of the family leave little time for leisure. What there is available is usually applied to the furthering of social or business success. But after the peak of activity in the forties, many people become aware of ill-defined uneasiness and a vague sense of frustration. Less absorbed by the drive to climb, they take stock of their lives and attempt a belated analysis of their objectives. Now, for the first time for many, comes the realization that, after all, their time is limited. Where has all their work led to? Ambition is prone to become vicarious, wishful thinking plans that the children may accomplish what the parents have left undone. Many men and women, unable to fulfill their own ambitious hopes, expect their sons and daughters to do infinitely better. Thus frequently more is expected of the next generation than its capabilities warrant. Boredom, unwelcome offspring of uselessness and frustration, begins its insidious undermining of morale and enthusiasm. These reactions are rarely recognized; there is only a consciousness of vague dissatisfaction, for mankind rarely has the courage to expose the reasons by honest thought. Innumerable rationalizations are introduced to gloss over personal shortcomings.

This ill-defined but nevertheless significant mental phenomenon of senescence comes earlier to some than to others. Activity, success in chosen career and continued responsibilities retard its appearance. The busy man avoids the necessity of thinking. In women, on the other hand, this reevaluation of late maturity frequently coincides with lessened physical responsibilities in the home and therefore greatly increased leisure, and also with the biologic endocrine turmoil of the climacteric. The efforts to escape take many forms, these are determined by the individual sense of values and previous cultural background. Women do not fritter their time away in "uplifting causes," clubs, teas, bridge contests, and theatrical matinees because they want to, but in desperate attempts to escape boredom. They are not happy thus. The tragedy lies in that such frantic and useless fluttering is unnecessary.

Partial but prolonged invalidism, particularly from the chronic and progressive disorders of later years, may bring about rather abrupt increments

in leisure. Not actually ill, but subject to distinct limitation in activity, are hundreds of thousands of middle-aged and elderly persons. Their enforced leisure can be a precious gift to swell the richness of their lives or it can be a sore trial. Existence too often becomes a pathetic attempt to "kill time." Weaker personalities react by hypochondriac depression or neurasthenic emphasis of every minor symptom. Stronger personalities bitterly resent their physical limitations and chafe and fret to their own detriment. These problems arise, however, only because of lack of preparation. They can and should be anticipated by every physician dealing with senescent patients.

Leisure affords opportunity for valuable physical exercise, beneficial to somatic structures directly and to the nervous system indirectly through mental relaxation and sounder, more refreshing sleep. The sedentary artificiality of modern urban existence makes for insufficient physical activity. This contributes to softness and obesity. Aging is too often used as an excuse for indolence. This does *not* mean that the soft middle-aged executive or clerk should rush from the thralldom of the desk into violent handball or tennis. Such is worse than folly. Danger lurks in the periodic debauch of violent physical activity for the sedentary business man so commonly indulged in during vacation periods or over week-ends. There are many kinds of fitness and muscular fitness is but one of them. There is no more need for a desk worker to be a giant of muscular strength, the Big Muscle Men of health clubs notwithstanding, than for a tractor motor in a small runabout. *Exercise must be appropriate to the individual*, particularly in the later years.

Exercise should be fun. Mental relaxation is one of the desiderata. The ritual of early morning setting-up exercises usually becomes a ludicrous performance. What sports are appropriate after the fourth or fifth decade? It is not necessary to limit the senescent to "old men's games." The crux of the question is *not so much what is done, but how it is carried out*. Even the patient with severe heart disease *can* climb stairs safely, if he does *slowly enough*. I knew a golf addict of seventy-seven who refused to let his angina pectoris dominate his life; he hired an extra caddy to push him up the hills. Many may continue their favorite sports far beyond the rocking chair age if they will but recognize their limitations and act accordingly. Honesty in self appraisal is, unfortunately, exceptional.

The capacity to tolerate and respond to violent and quick physical exertion depreciates with aging. Therefore those sports in which the participant's activity depends directly upon the acts of his adversary are best avoided in the years of later maturity. Tennis, handball, and badminton are examples of such competitive sports undesirable after the peak of maturity. Golf, archery,

capacity for exertion varies, these activities may be made strenuous or not as the occasion allows. By varying the dose of exertion to fit changing capacities, the therapeutic value of exercise is greatly enhanced.

Physical disability, however, may preclude any such investment of leisure as discussed above. What about the "shut ins" and their superabundance of leisure? Early encouragement to acquire hobbies appropriate to the personality and expected physical capacity of the patient can help immensely to prevent hopeless, helpless boredom and convert the enforced leisure into precious hours. Hobbies may be briefly classified as creative or noncreative. It is by creating something that we obtain our greatest satisfactions. *Recreation* is after all, derived from *creation*. It is relatively immaterial just what is created. It need not be physical at all, the satisfaction of curiosity brings new and creative ideas. Music, drawing, painting, modeling, writing, collecting and tending flowers are all feasible for the invalided. The physician must not forget that it is much harder to start such activities late in life (it is *not* impossible however, enthusiasm is contagious), and therefore he should encourage all patients to begin creative hobbies during or before maturity. If there is no mental preparation for senescence, senility is truly sterile. All productivity is not biologic.

Future Plans The aging and/or aged patient is often confronted by a fork in the road and uncertain as to which road to follow in the future. Retirement poses serious problems. Consideration of physical capacities and emotional and intellectual limitations may be invaluable in advising regarding the feasibility of the various possible plans for the future. These should be discussed freely and frankly, if the patient does not volunteer a statement of anxiety over the uncertainty of the future, the question should be raised by the physician. To do so often unlocks the gates and a whole flood of anxieties and problems requiring professional guidance are released.

The problem of residence of the aged is a frequent one and often difficult of solution. Few indeed are the households that can successfully contain two adult generations,¹⁵ and when senility advances to infirmity, the burden upon children and grandchildren becomes nearly intolerable. Facilities for the institutional housing of the aged and infirm are notoriously inadequate and unsatisfactory. There is urgent need for extensive study and energetic action in this area of the sociologic aspects of gerontology which comes so close to clinical geriatric medicine. Each patient presents an individual problem and generalizations are hazardous. There is ample room for argument as to the pros and cons of urging that the senile be kept at home or given residence in homes for the aged. Here more than one person is involved, it is a family and a community problem. Most older people are happier with those of their own generation and under the care of others than their children, for they develop a growing sense of insecurity and paranoid suspicion of those theoretically nearest and dearest.¹⁶ (See Chapter 7.)

Questions Last, but not least, the patient deserves an opportunity to ask questions. This may require considerable time and try the patience of the physician, but it is nevertheless an essential part of an effective health consultation. If the previous discussion, explanations, and advice have been adequate, questions should be unnecessary. The final word should be reassuring, for the aging and aged are often afraid. The promise of control, personal effort, and continued cooperation should be stressed, and tentative arrangements made for follow up. The most desirable frequency of recheck consultations will vary with the individual patient, but the maximum interval, if everything is found nearly optimal, should not exceed one year.

CHAPTER 7

MEDICAL CARE OF THE NORMAL AGED

FREDERIC D. ZEMAN

INTRODUCTION

WHEN G. Stanley Hall, the pioneer American psychologist retired as president of Clark University at the age of seventy-one years, he sought to take physical inventory of himself by consulting physicians regarding his health and the best means of preserving it. The results of this hygienic survey were most disappointing, and he was forced to the conclusion that medical men understood but little of the problems of old age.¹ In the twenty-five years that have passed since Professor Hall was put off with commonplaces and generalities, aging men and women have continued to seek expert guidance, often embarrassing their medical advisers with questions to which there were no adequate answers. Today medical investigators at the bedside and in the laboratory, in association with biologists and sociologists, are concentrating on the nature of the aging process, its concomitant diseases, and the resulting social complications. Some of the puzzles that confronted the last generation have become less confusing, and the present day practitioner is actually in a position to offer his older clients well thought out counsel according to their individual needs, based on scientific grounds.

The much quoted aphorism of Piersol and Bortz, "It is for science not only to add years to life, but, more important, to add life to the years,"² sets a high standard of achievement for us, and it is clearly not one to be attained by routine prescription of cathartics, diets, or sedatives, nor by perfunctory pats on senescent scapulae.

GENERAL PRINCIPLES

The physician engaged in treating old people requires specialized medical knowledge, but, in addition, must have abundant sympathy and understanding. Dr. Alfred Worcester, sage author of the classic, "Care of the Aged, the Dying and the Dead,"³ which is required reading for medical students and physicians, makes an instructive comment: "In the past I have maintained that a young woman's fitness for the nurse's calling can be determined by the reaction of babies to her care of them. In like manner I maintain that a young physician's fitness can be gauged by the reaction of his aged patients as it is evoked in his care of them. No surer measure can be had of his tact and courtesy, of his sympathy and devotion. These are indispensable qualifications of the physician. Some seem to have them as natural characteristics, but what such fortunate ones really have is rather the facility of expression, and this facility of bringing one's kind feelings into action can be acquired by practice."

William H. Mathews, outstanding pioneer in the solution of the social

problems of the aged, expressed this same thought from a different point of view "If I were giving a Civil Service examination to persons who were to have the care of old people I should first try to find out whether they liked old people. If I discovered one who did not I should mark her so low that she would never be appointed. I did not mean by that I expected them to like equally every old person they met. I did mean that they must not have the attitude of mind which some people have that old age is a sort of chattering nuisance to be shoved around and kept out of one's way. People who do not like old folks, just because they are old, ought to stay away from them. In so doing they will save both themselves and the old people unhappiness."⁴

For those who follow the precepts of Worcester and Mathews the rewards are unending, for old people with few exceptions are ideal patients. They are anxious to cooperate with the physician, they appreciate his interest in them and enjoy spreading his fame. The older patient must be carefully studied as an individual in relation to his family, his business and other interests, together with his ailments and physical disabilities, nor should the physician ever make the mistake of attempting to be facetious about matters the patient considers of serious import. Pleasantries often serve to lighten dark moods, but an ill timed joke may be poor psychotherapy. On the other hand, old patients like to be told the difference between chronologic and physiologic age, that one is not measured by birthdays but rather by what he can do. George Bernard Shaw, on the eve of his ninety second birthday, has advocated the abolition of birthday celebrations, and the many who have also suffered will gladly second his plea.

A physician's visit is an important event in the life of lonely men and women. It cannot be hurried without losing much of its value. The patient does not often have a sympathetic ear into which to unload his problems. The physician who knows how to draw out the interesting experiences of his patients who have gone through life with their eyes open is sure to acquire worthwhile knowledge bearing on the strange ways of the human race. At the same time he wins devoted friends.

Respect for long established ways of living, habits of thought and behavior must be faithfully observed. In the zeal to do good a physician may unwittingly initiate a chain of mental and physical alterations leading to actual illness. Probably the worst thing that can happen to older men and women is to be uprooted—taken from their old homes and familiar possessions to live with a married son or daughter. Since such a radical step usually results from loss of financial security, great tact is required on the part of the family to overcome the feelings of isolation, frustration, and inferiority that afflict the old people in these situations.

The commonly encountered pessimism of old people about their infirmities must be combated actively by the physician. Older patients are apt to regard their sufferings as the inevitable accompaniment and result of their years, saying apologetically, "Of course you can't do anything about that, Doctor, because I am too old." A careful history will bring out symptoms that the patient has almost forgotten, so carefully has he explained them away. By encouraging the patient to allow his doctor to judge whether any thing can be done, it is possible to elicit early symptoms and signs of serious diseases, and also to discover minor remediable conditions that have been the source of great annoyance and discomfort. False modesty on the part of

elderly women may also obscure significant symptoms, and on occasion even

many disorders affecting the
 the highest diagnostic acumen on the part of the physician. Old
 folks rarely have only one malady, and their sicknesses have a way of resisting
 therapy and lasting for a long time. The present writer has summed up these
 clinical peculiarities for his students in this sentence: Disease in the aged
 is characterized by multiplicity, chronicity, and duplicity. The physician
 must make every effort to avoid the stereotyped diagnoses so commonly
 applied to older patients. All heart disease is not due to arteriosclerosis. Very
 rarely it may even be congenital in origin, more commonly rheumatic or
 syphilitic in nature. The possibility of subacute bacterial endocarditis in
 cardinals must always be considered in the aged.
 ages, and thyrotoxic

Every bone pain in the aged is not caused by arthritis. Metastatic
 tumors, Paget's disease, osteoporosis, multiple myeloma also occur in these
 patients. Every attack of hemiplegia is not due to cerebral thrombosis. Neo-
 plasms, primary and metastatic, may cause focal lesions. Subdural hematoma
 may simulate an arteriosclerotic lesion and is curable by operation, but first
 must be diagnosed.

Although the aim of the physician is to make his knowledge available to
 all, Sleglitz has emphasized a special and particularly worthwhile phase of
 our problem. "The posts of greatest responsibility requiring the highest judg-
 ment, technical training and wisdom are held by older men. And with good
 reason. The

responsibility of preventive geriatrics."

RATING THE FUNCTIONAL CAPACITY

Physicians working with the aged have long realized that the detailed
 list of diagnoses that result from a careful health inventory of an
 aged person

work (See
 was develop

employed for over ten years

Class A Individuals capable of unlimited and unsupervised activity, to
 be trusted to go about the city in safety

Class B Individuals capable of moderate activity, to be trusted in the
 neighborhood of the Home, who may require escort of younger persons for
 extended or tiring trips

Class C Individuals whose capabilities are limited and whose activities
 need both supervision and assistance, require escort on the street, practically
 house bound

Class D

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to think in terms of the *whole individual* rather than in terms of the diagnosed disease. It is not at all uncommon to find on the face sheets of the clinical records of the Home an enumeration such as this: "hypertension, arteriosclerotic heart disease, sclerosis of coronary arteries, anginal syndrome, sclerosis of peripheral blood vessels (legs), osteoarthritis of hands, lumbar spine and knees, hemorrhoids, psoriasis." To many physicians, and certainly to most medical social workers, this catalogue of maladies would indicate a seriously sick individual, and yet many of the Class B patients have all these diseases, often in association with diabetes mellitus.

When the physician has arrived at a functional estimate, he is then in a position to advise as to the placement of the patient, the type of care needed, and the amount of work that the patient should be allowed to do. It is becoming increasingly clear to those working with the aged that Class A cases do not need institutional care and are best cared for in the community, preferably in their own homes or in boarding homes. Placement in an institution is usually required for three of the five groups. Obviously, Class D cases need active medical and nursing care, whereas Class C and E cases may require only the assistance of trained attendants in dressing and moving about from one part of the Home to another.

The work prescription is a serious responsibility of the physician, and is directly related to the functional diagnosis rather than to the patient's own eagerness to run errands, work about the Home, or spend more time in occupational therapy classes. We do not subscribe to the policy of *laissez faire* advocated by some on the ground that anything that makes an old person happy is, of itself, good for him. We believe rather that it is our duty to postpone as far as possible the inevitable cerebral hemorrhage, and to stave off the impending cardiac breakdown by the limitation of obviously harmful activity. Every now and then an obsessive, compulsive type of resident forces us to compromise with our ideals in the interests of peace, usually to our later regret.

From the standpoint of the administrative staff of a home the functional rating has definite practical value. This is equally true of the social service and the occupational therapy departments, all of which are staffed by lay workers, to whom many of the medical diagnoses, although familiar, are difficult to interpret. In these days of labor shortages the executive heads of institutions for the aged are more and more forced to turn to their residents for help in the actual work of their small communities. It is far easier to fit these old people to their tasks when an estimate of their capacity is at hand for guidance.

The social service department of a modern home for the aged has at least three major functions, *i e.*, giving advice to applicants for admission and to their relatives, the investigation of prospective candidates for admission,

greatly by the functional rating.

Today most of the progressive institutions have occupational therapy programs under the direction of trained workers. While any activity of an old

series and often producing useful objects for the institution or for sale. The director of the therapy department must be largely guided by the physician's functional diagnosis to which numerical ratings of the patient's manual skills may be added.

As the use of functional designations becomes more widespread, their practical value will become apparent to workers in family welfare agencies, in visiting homemaker organizations, and in state and municipal agencies caring for the aged. Medical diagnosis alone is not enough on which to base important decisions as to the type of care needed or the amount of financial assistance required, nor does it make clear whether the home surroundings are proper for the patient's needs.

The functional classification herewith presented has its application also in problems presented by industry. It would serve to weed out the unemployables, Classes C, D, and E, and would furnish the basis for judgment on the possible employable persons in Classes A and B. In large plants with well organized medical departments there is but little chance of old people venturing beyond their depth in seeking employment. Those physically or mentally unfitted are screened out by the examiners. There were, however, during the war many old men working as apartment house elevator operators who not only appeared feeble, but whose senile memory made it impossible for them to retain floor numbers for even a few seconds. It is clear that physical examinations and functional estimations would prevent these individuals from suffering humiliation and frustration.

The result of the carefully conducted inventory leads to the discovery of the patient's pathologic lesions and interprets them in terms of his ability to function. As has been pointed out above for institutional work, this same kind of estimate also forms the basis for the counsel we give the patient in our offices, relating to how much he should work, exercise, play, sleep, eat, drink, smoke, where he should live, how he should care for his teeth, his skin, his bowels, how to prevent specific diseases and finally, in spite of all this well intentioned advice, how to make life's later years a worthwhile and enjoyable experience.

WORK

A useful, interesting, and preferably gainful occupation is essential for the physical and mental well being of the old. In this respect environmental differences are important since the older man on a farm can be kept busy with chores which are progressively lightened as his capacity diminishes. On the contrary, older city dwellers, often retired office or factory workers, have little to do in the hours formerly filled with activity. Older women have an advantage over men because they can be useful in a variety of household duties geared to their strength and skills.

As previously indicated the work prescription requires careful consideration in order to prevent undue stresses and excessive fatigue. Use of the functional rating will help the physician and his coworkers to understand the needs of each individual. Pressing social and economic considerations make it necessary to utilize the energies of the older worker in peace time as in war time. The experience of the great industrial corporations is particularly instructive.

In 1942 Wharton described the work of the "old man's department" in the Dodge plant of the Chrysler Corporation, where the abilities of men handi-

capped by age or disability or both are utilized to the advantage both of the company and the men.⁸ The late Edsel Ford in an article entitled "Why We Employ Aged and Handicapped Workers" stated that of the workers employed in the River Rouge industrial area, more than 27 per cent were over fifty years of age, hundreds were over seventy, and seven workers were in their eighties. The psychologic advantages of keeping men gainfully employed are emphasized as a means of preventing the hopelessness so common in old men.⁹ The entire problem of the older worker has been admirably summed up by Carlson.¹⁰

Recent publications have shown the growing interest on the part of physicians, especially those who examine factory workers, in setting up standards and methods of studying the ability of special groups of the handicapped. Ritter has studied hypertension in industry and set up a yardstick for judging employability in cases of elevated blood pressure.¹¹ Poole and Bent,¹² as well as Kresky and Goldwater,¹³ have studied the employment potentialities of cardiac patients. Mosenthal has urged greater opportunities for diabetics in industry.¹⁴

The development of recreational centers for old people, the use of occupational therapy, and the role of hobbies will be taken up in more detail in the section devoted to mental hygiene. At this point, however, we wish to register a strong protest at the common business practice of retiring men and women at a fixed age, without consideration for their functional capacity, or their desire to continue in their occupations. The adjustment of these individuals to the new pattern of life sets up tensions that not infrequently lead to serious mental and physical disorders.

PHYSICAL REGIME

Exercise. Whereas work generally involves some degree of physical activity and is usually carried on to earn a livelihood, exercise is physical activity indulged in for promoting health and experiencing pleasure. The recent discussions on the ill effects of bed rest, by both medical men and surgeons, emphasize the necessity for some degree of activity of the entire body. To what extent the physician permits or advises the patient to exercise, will depend in large measure on his general physical status as revealed in the health inventory, and the resulting functional capacity rating.

Of course, any exercise which produces undue fatigue or uncomfortable symptoms referable to cardiac strain must be forbidden. Previous training and skill, however, make it possible for older men and women to carry on activities which would at first seem too much for them. The opinion of an old trainer of prize fighters comes to mind, "Horseback riding is only good exercise when you are learning." In city parks and on country bridle paths one sees old men sitting their horses surely and as comfortably as in an easy chair, apparently without effort. One riding master was still actively engaged in teaching young people in Central Park, New York, when he himself was in the early eighties.

In general, exercise is often made a fetish by physical training enthusiasts and overindulgence may be harmful. In view of the crowded, hurried life led by most men in the middle years, relaxation and rest are of far greater value. The physician will do well to explain that slow deliberate livers are more

apt to be long livers. Proper cultivation of leisure and judiciously practiced laziness conduce to longevity.

Diet. Planning the dietary of the aged individual requires careful consideration of many factors. Not only must the adequate number of calories derived from the proper percentages of protein, fat, and carbohydrate be furnished, but also sufficient concentrations of minerals and vitamins. In addition, psychic factors underlying likes and dislikes, dietary customs based on religion, and the actual consistency and appearance of the foods served must be borne in mind constantly by the practicing physician and by the institutional dietitian. They must never forget the fundamental problem of cost, and endeavor to fit the diet to the patient's means.

The work of Sherman on the effect of high Vitamin A intake in promoting rat longevity¹⁵ has focused our attention sharply on the therapeutic value of planned diets in the aged. Sherman points out, "The previous general progress of public health had increased the life expectancy of the infant but not that of the grown person. Now the nutritional improvement of the norm raises the life expectation of the adult as well. The extra years thus offered are not to be pictured as added to old age. Rather it appears that something like an extra decade can be inserted at the prime or apex of life lived in accordance with today's newer knowledge of nutrition. Life becomes longer because it is lived on a higher health level throughout. The apex of attainment is higher, the period of prime is longer and in human terms, there is a smaller percentage of years of dependence."

McCay has shown that diet can retard the aging process and that types of diet may actually lengthen the life line of rats.¹⁶ By feeding these animals a diet containing all needed substances for normal growth, but low in calories, the average life span has been increased significantly and the development of inflammatory, neoplastic, and degenerative diseases common to this species in old age definitely retarded.

Proving the value of undernutrition in rats is of extreme interest in connection with well established bad effects of overweight on life expectancy in human beings. It is likewise a striking corroboration, nearly four centuries later, of the views of Cornaro, the famous Venetian exponent of extreme moderation in eating.¹⁷ All of the foregoing assumes even greater significance in view of the importance now ascribed to cholesterol in the pathogenesis of arteriosclerosis. The present day tendency is to reduce fat intake in order to prevent or to limit the deposition of cholesterol in the intimal layers of the arteries. Of interest too is the fact that the National Research Council's Food and Nutrition Board recommends that the diet in general should contain 20 to 25 per cent of its calories in fat. Among Eastern peoples often less than 10 per cent of the diet is in fat and Snapper has emphasized the rarity of arteriosclerosis among the inhabitants of North China.¹⁸

Since there is a decrease in muscular activity and slowing of body processes in old age, the total caloric requirement of old people is less. "It is roughly estimated that the decrease in food requirement due to old age from the total fuel which would be required by an adult of the same degree of activity, is about 10 per cent between the ages of sixty years and seventy years, about 20 per cent between the ages of seventy and eighty years, and about 30 per cent after that."¹⁹ This rule may be used in calculating diets for older patients.

The significance of protein in the diet in health and disease has received great emphasis in recent years. In addition to furnishing large quantities of the vitamin B complex and minerals to the diet, protein foods play an important role in maintaining plasma proteins and resistance to infection.²⁰ While the exact requirements of protein are not known, the safest plan is to follow Sherman's rule of 1 gm. of protein per kilogram of body weight as a minimum requirement. Stare and Davidson point out that defective or absent teeth, achlorhydria, and chronic constipation may lead to anorexia and low protein consumption, and warn against allowing an old person's distaste for meat to bring about a dangerously low intake.²¹ About 10 per cent of the total calories should be in protein. During disease and convalescence an even greater protein utilization is needed.

In view of our present day insistence on the harmfulness of fats it is wise to limit the fat to about 10 to 15 per cent of total caloric intake. Starches and sugar will make up the balance. The inclusion of milk for its food value and its calcium and phosphorus content is obligatory. Fresh fruits, salads, and freshly cooked vegetables add minerals and vitamins to the diet. In this connection it must be stressed that healthy old men and women consuming adequate diets do not need vitamin supplements. Ruffin's summary of the use and abuse of vitamins may well be borne in mind by the physician caring for the aged.

"1 Vitamin therapy is definitely indicated in patients having objective evidence of a deficiency state, but should always supplement dietary treatment, never replace it.

"2 Vitamin therapy is useful in preventing the development of secondary deficiencies in chronic wasting diseases and in pre- and post operative care.

"3 In the absence of organic disease the individual who consumes a diet, adequate in calories, consisting of fruits, milk, eggs, a variety of meats, and green vegetables does not need additional vitamins.

"4 Vague symptoms such as weakness, fatigability, insomnia, nervousness and irritability, are more apt to be due to overwork, nervous tensions, or to social, domestic or financial difficulties, than to a vitamin deficiency.

"5 For the most part, prolonged vitamin therapy in the absence of obvious disease is useless."²²

Sadow's discussion of diets for the elderly is practical and of especial value to the institutional dietitian. She points out that "a dietary routine of three light meals a day, with midmorning and mid afternoon feedings usually tends to cut down the total quantity of food eaten and is more satisfying. Long waits have a tendency to make elderly people uncomfortable, especially the

too the narrow margin between the therapeutic dose and the toxic dose. Many older men and women do not like the taste of alcoholic beverages

and do not enjoy the effect. On the other hand, for those who have been in the habit of moderate indulgence there can be no serious objection to its use in the absence of gastric disorders. Of late alcohol has been recommended freely by cardiologists for angina pectoris, coronary sclerosis and arteriosclerosis obliterans because of its vasodilating action. Here too it is appreciated by the habitual drinker and found distasteful by others.

Physicians must remember that habituation to alcohol does occur in later life and must be wary in prescribing it. The present writer has known two charming old ladies who regularly drank themselves to sleep every night with a tumblerful of sherry!

Tobacco In spite of increasing consumption throughout the country and in spite of national advertising campaigns making much of the number of physicians who favor special brands, the evidence is slowly accumulating that tobacco is harmful to many. While the concept of the "tobacco heart" has not been accepted, smoking has definite cardiovascular effects, some of which are certainly harmful in conditions such as peripheral vascular disease. In addition some authors believe that the increase in the incidence of pulmonary carcinoma in the past thirty years is due in part, if not wholly, to the increased use of tobacco.

As with alcohol, moderation is the only safe method of indulgence. Sometimes the agony, real or imagined, of foregoing smoking will do the smoker more harm than the noxious fumes themselves. It is often possible to persuade patients of the benefits of nonsmoking by having them abstain totally for a week or two and then leaving it to their judgment as to whether or not actual benefit has resulted. Occasionally one encounters individuals in whom nasal and throat irritation as well as indigestion can be ascribed to cigars or cigarettes.

Sleep Perhaps the most constant functional change associated with the aging process is increased fatigability, which is to be ascribed to a summation of the anatomic and physiologic alterations peculiar to senescence. Aging individuals should be assured that their increased need for rest is not in itself pathologic, and that they should not be ashamed to lie down for a nap after lunch or before the evening meal.

Insomnia is common in the old, and requires careful study before successful treatment can be instituted. As with younger persons, fears, worries, and anxieties carried to bed effectively prevent relaxation. Open discussion of these mental factors may help to dispel them. The bedroom should be dark, the bed comfortable, the bedclothes light but warm. A warm milk drink before retiring or a hot water bottle at the feet may help induce sleep. Men or women with irritable bladders should be warned against consuming fluids between the evening meal and bedtime. Often being awakened by the desire to void, the patient is unable to get back to sleep. Coffee or tea at the evening meal is best forbidden to light sleepers.

The physician must carefully analyze the patient's habits and behavior, inspect the bedroom if necessary, and use every stratagem to avoid the prescribing of drugs. When all else fails medication must be prescribed, for a sleepless night may be more harmful than a weak barbiturate. Sometimes drugs work more by suggestion than by their specific action, since many an elderly person will sleep well on phenobarbital, $\frac{1}{4}$ grain at bedtime. Ordinarily this drug is not used as a sleep producer because of its slow absorption.

Soluble phenobarbital is preferable. When stronger barbiturates are needed, those rapidly absorbed and excreted, such as sodium pentothal, $\frac{3}{4}$ to $1\frac{1}{2}$ grains, are most effective. So much has been written of late in newspapers and popular magazines about the harmfulness of barbiturates that patients need to be told that in moderation and under a physician's guidance they may be taken in safety.

Care of the Bowels. The physician who is well informed on the proper management of the bowels will give his older patients more satisfaction than one who is highly trained in the fine points of cellular metabolism and enzyme chemistry. The prevention and treatment of constipation is of never ending interest to most individuals, and the patients' complaints about this disturbance cannot be dismissed lightly without overlooking possibly serious conditions. In any patient a sudden or abrupt change in bowel habits is of great significance, as is rectal bleeding, the source of which must be checked carefully. External inspection, digital examination, anoscopic and sigmoidoscopic investigation and x-ray studies will usually disclose the cause. Occasionally ulcerations of sigmoidal diverticula in old men will cause profuse rectal bleeding.

Functional causes of constipation are common in old people, such as sedentary habits, lack of exercise, irregular times of going to stool, inadequate water intake, deficiencies of bulk and vitamins in the diet, weakened abdominal and intestinal musculature, and psychic factors. The presence of these conditions will be apparent from a careful history. Often it is necessary to explain to the patient that skipping a day is not of serious import and that a large amount of material need not result from each evacuation.

The use of drugs must be carefully safeguarded, and every effort made to persuade the individual to try nonmedicinal aids such as increased water intake, proper diet, proper habits, and light exercise. Detailed explanations by the physician of the rationale of these procedures will often be rewarded by therapeutic results, startling most of all to the patient.

For years the most commonly described medicinal agents have been cascara, phenolphthalein, and mineral oil. Of these only cascara, either in tablet or liquid form, remains in favor. Phenolphthalein has a tendency to cause skin eruptions, and is apt to be uncertain in action. Mineral oil not only commonly leaks from the rectum, causing great inconvenience, but interferes with digestion and vitamin absorption. Mechanical laxatives seem preferable because they furnish smooth bulk and lubrication without stimulation, irritation, or absorption. The following gums are in common use: agar agar, karaya, sterculia gum and ground psyllium seeds. Bran is not advised because it tends to form impacted masses in the sigmoid and rectum. Cascara may be added to gum laxatives when indicated, as in "bassorin" and "mucara." "Siblin" is a smooth bulk laxative containing vitamin B²⁴.

Enemata are valuable in the aged, particularly when digestive symptoms are thought to be due to laxative drugs. A pint of warm normal saline solution, or a glycerin enema ($\frac{1}{2}$ ounce of glycerin to 1 pint of water) may easily suffice when introduced slowly. Where defective musculature is present, it is

The proper cleansing of the perianal skin after stool is of paramount

importance in preventing skin irritation and infections in that area. Warm soapy water should be used and the skin patted dry. Often old people are unable to do this by themselves and need assistance. The softest tissues should be used and the skin protected afterward by rubbing in a small quantity of lanolin ointment. In cases where protruding hemorrhoids are present an astringent ointment containing a mild local anesthetic may be used to advantage.

Care of the Teeth. The loss of many or all of the teeth so commonly noted in elderly individuals is not to be looked upon as part of the aging process, but rather as the result of pathologic processes either untreated or unsuccessfully treated, as the result of accidental injuries, or of surgical removal in the course of the treatment of diseased teeth. The prevention of these conditions properly begins in childhood and modern dentistry can take pride in the progress that it has made in educating the public and in the results that it has attained with a small part of the total population. Spreading the advantages of thorough dental care to more and more people is one of the essential parts of the program of national health conservation.

The physician caring for old people must understand the problems of the dentist and be able to interpret to his patients the scope and limitations of the art of dentistry. Loss of teeth affects the function of mastication, produces structural changes in the jaws, brings about significant alteration in speech, especially as regards consonants, modifies the expression of ideas and emotions by the face, as well as the appearance of the face at rest. Along with these structural and functional changes go psychologic disturbances, most marked in individuals of narcissistic tendencies.

Horner's statement deserves the physician's careful consideration. "To the patient who has lost his teeth, the placing of an artificial denture is one of the important events of his life."²⁵ In discussing the education of the denture patient Horner believes that it is necessary to correct erroneous ideas derived from irresponsible sources, that only through understanding can intelligent, willing, and cheerful cooperation be achieved, and lastly that the dentures, unlike other dental work, are under the patient's control and can be worn or removed as the patient sees fit. His instructions to denture patients emphasize the need for patience in becoming accustomed to the new devices which are at best an imperfect substitute for natural teeth. He also points out that "the more advanced the age of one who is learning to wear artificial dentures, the harder will be the task of learning to wear them. A youth, aged eighteen years, may master the art in a week, whereas a man, aged eighty years, cannot be expected to acquire in the remaining years of his life anything like the proficiency that the youth might in the wearing of his dentures."²⁵

With the physician and the dentist working together more closely than is at present customary, we may expect far better results in the care of the teeth in old people. Underlying nutritional disturbances and local infectious conditions will be cleared up, to the end that the dentist may do his work under optimal conditions. Through understanding the possibilities of prosthetic dentistry the physician will be able to give real assistance in teaching the denture patient. In addition physician and dentist will join forces in the search for sources of chronic irritation in the form of rough jagged teeth, poorly fitting dentures, or projecting denture fittings.

Care of the Skin. The common occurrence in the elderly of a variety of skin disorders, for the most part secondary to the changes of senescence,

makes obligatory careful attention to measures designed to ward off serious consequences. In general the customary habits of cleanliness are adequate, such as frequent leisurely baths in warm water using a good quality unmedicated soap. Not infrequently older individuals for reasons of physical or mental origin neglect even these elementary practices. Sometimes incomplete drying of the skin leads to areas of irritation. Gentle handling and the use of mild soaps will insure against most discomforts. Occasionally so called "bath itch" is encountered which is due either to sensitivity to soap or too vigorous rubbing of the skin or both. Here the only cure is cessation of bathing and the use of soap for varying lengths of time. Use of the newer nonsoapy cleansing agents may be required.

Lessened resistance of the skin to infection is due to thinning of the layers and to poor blood supply. Slight scratches or cracks give entry to pathogenic organisms. Patients should exert great care in trimming their finger nails to avoid injuries which lead to the development of paronychia infections or felons. Hollander advises that old people do their own manicuring with instruments that have been boiled to ensure cleanliness.²⁶ These precautions must be redoubled in the care of the feet and the toenails.

Only expert podiatrists should trim the toenails of old people. Unsteady hands, poor vision, and stiffness in the joints prevent accurate manipulations. In institutional practice podiatrists visit twice weekly to ensure painstaking attention. In cases of diabetes mellitus or arteriosclerosis obliterans the slightest injury may lead to grave infections and gangrene. Modern clinics for diabetes and for peripheral vascular disease devote great attention to the education of their patients and also provide podiatrists for the care of the feet. Daily washing of the feet is advised. Woolen socks are recommended. Broad comfortable shoes are insisted upon to avoid pressure sores and calluses. Dry skin is to be treated by rubbing in cold cream, lanolin, or cocoa butter after bathing. At night the use of hot water bottles or electric heating pads is completely forbidden, and the use of bed socks is recommended. Only by strict adherence to these rules can be prevented the onset of infection, which in diabetics with peripheral arteriosclerosis may in many instances lead to major amputations.

In addition to the rules of hygiene and the measures to prevent infection the physician must bear in mind the constitutional conditions leading to skin disturbances and the variety of the drug eruptions which occur in the old as well as in the young. Above all he must watch carefully all the benign lesions of the skin such as warts, moles, nevi, or skin tags for any changes that might indicate a malignant metaplasia. Known precancerous lesions, such as senile keratosis and cutaneous horns, must always be subjected to closest scrutiny. Any lesion which crusts over repeatedly without healing is at once suspect and needs the attention of a dermatologist.²⁷

PREVENTIVE MEASURES

Prevention of Accidents In 1941, 15,900 deaths in the United States resulted from falls in the home, 80 per cent of which occurred in persons over 65 years of age. These accidents can be avoided if the physician makes a careful physical examination of his patients and takes into account the factors which may lead to falls. He should advise his patients to take precautions to avoid falls by studying the factors which lead to them and by making changes in his daily work in private

dwelling and in institutions, and by using every means in his power to educate board members, administrators, nurses, relatives, and patients

The first requisite is that physicians must become accident minded, and must always remember that accidents are the fourth most important cause of death at all ages, and that the aged by reason of their infirmities are particularly likely to suffer injury and death from falls and motor vehicle accidents. The forward looking physician will cooperate with private and governmental agencies in promoting safety campaigns. He will aid in efforts toward safer motoring by setting a good example himself.

In his dealings with his older patients he will endeavor to give them pains taking medical care, and in his examination will make every effort to detect ocular, auditory, cardiovascular, musculoskeletal, neurologic and mental abnormalities that may predispose to accidental injury. He will tactfully warn his aged client of the possibilities of danger, and if conditions warrant actually forbid certain kinds of activity such as walking at night or driving a motor car. At times the cooperation of the family will have to be sought in order to avoid serious danger. The education of the family in the matter of accident prevention is the doctor's duty.

Further, the physician must be on the lookout for danger in the patient's home environment. He must warn against small loose rugs, and advise that they be nailed down, and that the same should be done to larger carpet rugs with turned up edges. He must see that shaky stairs and rickety bannisters are repaired. Unsteady bedside tables should be removed, but above all an unobstructed pathway must be maintained from the bed to the bathroom, especially at night. A shielded bedside light kept burning all night is a helpful safety measure. The bedrooms and living rooms of many old people are cluttered with furniture with which they hate to part, with the result that one day the leg of an unnecessary table will lead to a fall and a fracture of the neck of the femur.

In hospitals the physician should conduct a continuous educational campaign to apprise nurses of the dangers of accidents and to teach them how they can help in prevention. He should not hesitate to order side rails where needed, especially at night, for patients who are disturbed or under the influence of drugs. He should also bear in mind the danger of overdosing old people with sedatives. He always remembers the fire hazards, particularly of institutions, and considers himself personally responsible for the removal of potential causes of fire, the installation of proper emergency equipment, and the instruction of personnel on their duties in case of fire. He should spare no energy in reminding boards of directors and administrative heads of their responsibilities to the old people.

In brief the physician must assume an aggressive attitude towards the whole problem of accidents and accident prevention. speak firmly and decisively when occasion demands, and put into actual practice the lessons of experience. Under his leadership an active program of prevention can be initiated and carried forward to the point where falling accident rates for old people will prove the value of his efforts.

Prevention of Infections The prevention of infectious diseases in the aged should be our aim at all times and with thought much can be accomplished by the practitioner. The following steps in such a program may be enumerated

- 1 The education of physicians in the appreciation of the capacities

well as the altered reactivity of the aged, thus inducing a more constructive attitude to the aged sick, both medically and surgically

2 The education of the patient in the care of the skin in general and of the feet in particular, as noted above

3 Adequate diets for the old in order to maintain protein, mineral, and vitamin levels, and thus promote resistance to infection

4 Early treatment of upper respiratory infections, including isolation of older persons from others suffering from head colds

5 More careful treatment of the aged diabetic There is a tendency to dismiss this type of case casually since usually it is mild Give the diabetic an infection, a fall, or even a mental shock, and the mild case may become a severe one overnight

6 The education of the public in the elimination of accident hazards which so often open the way to infections The physician, as he visits his old patients, has an important role to play, as is discussed in detail in the preceding section

7 Earlier surgical intervention in known gallbladder disease and in prostatic hypertrophy with obstruction will make it possible to operate when the patients are best able to endure the stresses, and before infection complicates the picture

8 Furtherance of research projects that will provide us with new methods for sustaining the waning defensive powers of the aged ²⁸

Prevention of New Growths. Throughout the preceding discussion emphasis has been placed on the early diagnosis of malignant tumors in various parts of the body While most authorities agree that cancer is never either a part or the result of the aging process, the statistical fact remains that it occurs with increasing frequency as age advances While the cause is not known, many predisposing factors are well understood and it is possible to remove sources of chronic irritation or known precancerous lesions when ever detected Particular reference has been made to lesions of the skin and mouth which are easily accessible to frequent inspection The educational campaigns designed to increase popular knowledge of the cancer problem will bring more and more of these patients to physicians who must know how to differentiate good from bad, and when in doubt seek expert advice and substantiation by biopsy

On the other hand the early diagnosis of visceral malignant new growths presents more difficult problems, only partially solved by x-ray examinations, by bronchoscopy, gastroscopy, sigmoidoscopy, and cystoscopy Too often these investigations are initiated when the tumor is already far advanced The work of St John and coworkers, cited above, in detecting by fluoroscopy three cases of gastric malignancy in over 2413 asymptomatic individuals, and the now widely accepted and practiced technic of routine chest examinations of children and adults, point to the value of routine x ray examinations even in the absence of symptoms Until the development of a specific blood test our diagnostic approach will have to be limited to the methods suggested

Physicians and surgeons have gradually come to realize that old people stand surgical intervention extremely well Thanks to modern methods of ante- and postoperative care, of anesthesia, and to the use of antibiotics, extensive operations are today more feasible in old people with a minimum of complications This has changed our attitude toward abdominal explora-

tion for the relief of supposedly inoperable tumors. Often great benefit can be given the patient and frequently, particularly in carcinoma of the large bowel, a real cure is achieved.

Prevention of Serious Complications in Certain Common Diseases. In discussing the prevention of infections mention has already been made of the value of early surgical intervention in cholelithiasis and in benign prostatic hypertrophy. When operation is undertaken while the patient is still in good condition and before infection has set in, the prognosis, both immediate and late, is extremely good.

The complications of diabetes have also been touched upon with reference to prevention of infection, and to the influence of infection upon the diabetic process itself. Diabetes in the aged often seems like a mild condition since the sugar tolerance is relatively high, but so many complicating associated conditions are present that the mild case may become a grave one with but little warning. The aged diabetic must be treated with the same care and attention to detail as is given to younger individuals. (See Chapter 14.)

In the management of heart disease of any variety in the aged, the physician's attention must be directed to the prevention of heart failure. In arterio-sclerotic heart disease the prevention of coronary insufficiency (anginal syndrome) and of recurrent myocardial infarction should also be his aim. This is to be accomplished by eliminating disturbing psychic factors where possible, by keeping down the body weight by regulating the amount of exercise taken, by limiting or forbidding the use of tobacco and the administration of vasodilator and sedative drugs. In patients threatened with congestive heart failure the water, salt and food intake as well as the amount of activity must be sharply limited. The influence of infections in precipitating acute heart failure must be recognized and the patients protected from the common cold and influenza. Where patients are on maintenance doses of digitalis they should be checked at frequent intervals, but also need to be taught how to take their own pulse while at rest to detect digitalis intoxication in its incipency.

In all old people with cardiac disease careful attention must be given to the hemoglobin and red cell values in the circulating blood. Chronic anemia may severely limit myocardial function and add to the difficulties of the overburdened heart. Often these anemias respond promptly to iron and liver therapy, with striking subjective and objective improvement. Acute anemia from profuse bleeding may induce myocardial infarction without coronary thrombosis. (See Chapters 24, 26 and 27.)

MENTAL HYGIENE

Grey hairs have many evils without end
The old man gathers what he dare not spend
While as for action do what he will
Tis all half hearted spiritless and chill
Inert irresolute his neck he cranes
Into the future grumbles and complains
Extols his own young days with peevish praise
But rates and censures these degenerate days 9

In these vivid lines the Roman poet described his elderly contemporaries and for all succeeding generations the portrait has lost none of its accuracy. In the last century, however, a steadily growing understanding of the mind in old age has led to extension of the mental hygiene movement to the later

years of life. We have ceased to blame the old for their peculiarities. The effort has been made to explain why old men grumble and complain, why they seem to have lost interest in the world, and why they lose confidence in themselves. While completely satisfying answers to all these questions are not available, enough insight has been gained to make possible a program for bringing more happiness, greater satisfaction, and less frustration into the lives of the old. We know today that the provision of economic security alone is not enough to achieve these ends, since the well-to-do must face reality as well as the indigent.

The problems which a mental hygiene program seeks to solve are three-fold—those arising from old age itself, those arising from the fact of dependency, and those arising from the make-up of the society in which we live. In addition many psychic maladjustments occurring in old age date back to childhood and adolescence and are not the result of either old age, dependency, or social change.

The aging process in the central nervous system is characterized by cellular atrophy and diminution in the actual number of cells, together with vascular changes not necessarily a part of a general arteriosclerosis. These anatomic changes lead to functional alterations such as lessened olfactory, visual, and auditory acuity, slowing of attention, memory, and learning ability, together with lessened speed of neuromuscular reactions. These changes come on slowly and at different speeds in different individuals, and are to be thought of in terms of partial rather than total losses. Psychologists have given a definite answer to the old query, "Can an old dog learn new tricks?" He most certainly can, if he wants to learn. (See Chapter 5.)

When the cerebrospinal changes of senescence are aggravated by frank vascular insufficiency, as is caused by cerebral arteriosclerosis, the ability of the mind to respond to stresses and strains of all kinds is seriously impaired. Thus severe nervous shocks, febrile illnesses, drug intoxications, surgical operations, nutritional deficiencies, and cardiac failure commonly give rise to psychotic manifestations, many of which are of a temporary nature. In the same way the minor annoyances of life, changes of scene and of habits, cannot easily be compensated for, and the old person develops anxieties, depressions, becomes sad and morose, and repels all efforts at assistance.

The fact of dependency is a bitter reality for the aged to grasp. From childhood onward we are taught to provide for our old age and savings are accumulated with the idea in mind that one day earning power will be gone. Due to the unpredictable nature of financial depressions savings go by the board and many a thrifty couple, through no fault of their own, find themselves in their sixties dependent either on their children or on the state. Cruel disillusionment is seen commonly in individuals admitted to homes for the aged which, no matter how homelike, friendly, and kind they may be, nevertheless represent the dreaded "poor house over the hill." Even in the midst of loving families tensions arise for the aged parent is reminded on every

Our present day civilization has brought about an extension of life, and at the same time has made it difficult for older people to follow gainful employment. Some exceptions to this rule, in the great industrial corporations,

have been mentioned above. To date, however, no serious effort has been made on a large scale to study the functional capacities and skills of older men and women in order that they be kept at work as long as possible. The war years brought many elderly men out of retirement, back to jobs and to the real happiness that comes from participating in the world's work. In many modern institutions the residents have not only the benefit of occupational therapy but also actually help in activities of the organization, in the kitchen, the laundry, the sickrooms, and the garden. The physician in city practice frequently notices how much happier the old women are than the old men. The latter have practically nothing to do while their mates busy themselves with cooking, housekeeping, and the care of young children.

Many of the difficulties of older people date back to personal maladjustments of long duration. The immature, the compulsive, the narcissistic, the psychopathic types, often attain old age without improvement, or the "mellowing" which age is popularly supposed to bring. The writer has had under his care a woman of seventy-five years who has always enjoyed the luxury of a personal maid, but has never been able to win the love of her own child. When the maid in combing her hair runs into a tangle and causes pain, the old lady often bites the maid's hand in uncontrollable rage. This persisting childhood trait cannot be blamed on old age. Of all the adjustments that life demands the realization of old age and the adaptation to it is the severest test of mental stability. It is not surprising that those who have previously had difficulty in meeting reality will have even greater trouble at this trying period.³⁰

The first requirement in a program of mental hygiene is education, for physicians, professional nurses, and social workers. These in turn will educate the old people themselves and their families. The medical man must supplement his sympathy with knowledge derived from books and from actual experience.³¹ He would do well to subscribe to one of the medical journals devoted to the problems of aging, and should miss no opportunity of attending lectures by authorities in the field. The physician also needs to be familiar with social problems as well as medical ones, and to be interested in the community, and in the general public.

He must cooperate with the community, and with the general public, and must be familiar with the needs of the aged.

The nurse must have an extensive literature in geriatric nursing to cover but must give her aged patients the benefit of what she has been taught to do for the young. She will find that these attentions will come to her old patients as something of a surprise, since they are so accustomed to being cast aside.³² As Miss Marsh says regarding the chronically ill and the mentally ill, "It is a lonely existence at best and must be brightened by cheerful companions and a staff with a sense of humor."³³

Social workers must understand the basic principles involved in the medical and nursing care of the aged and in addition must be familiar with the

should likewise be the fountainheads of knowledge on the Federal Social Security Law and the privileges it accords the aged.³⁴

The concerted expert efforts of this group will inspire the family and the patient with confidence to follow guidance. An example must be set to the patient of sympathy and understanding tempered with firmness and free from maudlin emotion. The family must be taught that the old man or woman is an individual who can be helped to happiness by intelligent understanding, a human being with certain fundamental likes and dislikes, a once potent member of the social group, who cherishes privacy and independence, who is most satisfied when helping ever so little toward the family welfare, and who still wants to be respected and as always, craves true love.

In institutional work mental hygiene programs and recreation programs are apt to be thought equivalent. Actually recreation is only one part of the whole effort toward promoting psychic well being. The well tested practices at the Home for Aged and Infirm Hebrews deserve description in some detail. Here the entire staff is instructed that the residents are to be treated with respect and sympathetic understanding. Rigid adherence to formal rules is avoided, every attention is paid to providing good looking clothing, and study is devoted to finding out particular interests as well as likes and dislikes. The newcomer is greeted by a committee of residents who take him around, show him the lay out of the building, and explain its customs and usages. The Home Club is an organization of the residents, replete with officers and committees. One group visits the sick in the infirmary, another keeps track of birthdays, and presents the greetings of all to the "birthday child" at the noonday meal. Still another consults with the Executive Director on matters involving infractions of the rules or makes suggestions to him for innovations. The Home News, a periodical brought out entirely by residents, gives a formal outlet for artistic and literary gifts. After the newcomer has become acclimated, the Social Service Department, in consultation with the physicians, makes an assignment to some form of work in the home or to activity in the Occupational Therapy Department.

A well equipped Occupational Therapy Department, under the guidance of a registered therapist, gives opportunities for weaving, sewing, rug making, embroidery, raffia work, book binding, wood carving, metal work and carpentry in many forms. Bedridden patients are visited in the infirmary and appropriate handicraft activity assigned. Many of the articles made are salable, but the true object of the work is to give the patient satisfaction rather than attain a commercial output.

The recreation program is under a trained director and includes weekly up-to-date movies, entertainments by visiting professionals and amateurs, bingo games, dances on festive occasions, appropriate holiday exercises, and plays put on and acted by the residents themselves, largely of their own choice. Older staff members still recall a "Floradora Sextette" in which each "pretty maiden" was over seventy years of age. These entertainments, as well as the Home Club meetings, give our guests a chance to make new friends in their own age group with similar interests, and also provide the company of the opposite sex.

The residents' families and friends are urged to pay frequent visits, and a flexible schedule of visiting hours makes this easier of accomplishment. For the many without relatives, a panel of volunteer visitors spends several hours two or three times weekly with groups in the dormitories and infirmary wing. The work of the dietitian in providing dainty, attractive meals is of

great significance in promoting mental good health on the gastronomic side. A large library and reading room provided with daily papers in several languages and current magazines is well patronized by the residents. Religious services are held twice daily in an attractive synagogue.

As a result of this comprehensive program the most remarkable results are achieved in rehabilitating the old people. Downcast and depressed on entrance, their broken spirits revive under the friendly efforts of their fellows and of the staff. In the Home they find security, and an opportunity to build a new life. We enjoy watching the development of initiative and leadership in these people who had entirely lost hope.

The example of the work of the Home helped to stimulate the establishment of recreational centers for the aged in New York City. Here the old man or woman living alone or in a crowded home finds a haven, where friendship and understanding await him, where he can read, model, paint, play cards, or just sit and smoke in peace. Sheltered workshops give the able bodied old man the opportunity for useful work that industry has perhaps denied him.

The experience of institutions and public welfare agencies can teach the practicing physician how to help his older patients.³⁵ He must encourage hobbies of all kinds, outdoor as well as indoor, and insist upon the value of social contacts, of reading, of listening to the radio, and of attending lectures as well as religious services. The aid of clergymen cannot be overestimated, since nothing sustains like true faith and no comfort can rival that offered by religion. Pastoral counselling and guidance must take their place along with the efforts of the social worker and the physician in caring for old people.³⁶ The pastor may be of great value in interpreting the older patient to his own family.

To use bibliotherapy, the physician must himself know the books that are worth while, and by his enthusiasm kindle interest. The Holy Bible, Cicero's "De Senectute," Cornaro's "Sober Life," Shakespeare's "King Lear," his other plays and sonnets, and Montaigne's "Essays," Bacon's "Essays," the writings of Oliver Wendell Holmes, and the poems of Tennyson

Lawton's "Aging Successfully," should also be put in the hands of the patients.³⁷

A common problem of older individuals is the question of when to retire from business or profession. Compulsory retirement at a fixed age is surely a pernicious practice since it often forces men who are young for their years into unwanted idleness. Retirement should be based on functional

into a Promised Land, where they can enjoy the hobbies, music, and the arts, as well as travel, long deferred for want of time

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CHAPTER 8

PRINCIPLES OF GERIATRIC SURGERY

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SURGERY in the aged is constantly assuming greater importance as a result of the growing number of elderly persons in the United States. The census figures from 1870 to 1940 and the estimates for 1946 and 1980 illustrate the increase.

POPULATION (BUREAU OF CENSUS)

	Total	Number 65 Years and Over	Per Cent
1870	38,558,371	1,156,750	3.0
1900	75,994,575	3,115,770	4.1
1940	131,669,275	8,953,510	6.8
1946	141,228,693 (estimated)	11,014,840	7.8
1980	163,677,000 (estimated)	18,986,530	11.6

Between 1870 and 1940, the total population increased 3.4 times, but the age group 65 and over increased 7.7 times. Between 1930 and 1940, the total population increased 7.2 per cent, while the group 65 and over increased 35 per cent, roughly five times as rapidly as the general population. Another catastrophic war would probably augment this trend. Greater longevity will bring an increasing number of elderly patients to the surgeon.

GENERAL PRINCIPLES

The elderly patient is subject to all the surgical ailments common to earlier life. A gangrenous appendix or a strangulated hernia can occur as well at seventy as at thirty-five, and the indications for surgical intervention are the

age confers resistance and immunity to many infections that have been encountered and conquered earlier in life.

The treatment of malignant lesions constitutes a large proportion of surgery in the elderly patient. Such surgery is frequently extensive and shocking, but adequate preparation, a well planned and gently executed operation, and intelligent after-care should offset the handicap of old age. The fact that cancer grows more slowly in the aged offers a better chance for cure and is an incentive for performing a radical rather than a palliative operation. Obviously this does not preclude palliative procedures which are justified for the alleviation of pain and the relief of symptoms where radical surgery would entail too great risk. Age as a factor in determining either surgical risk or the type of operation to be carried out is steadily becoming less important.

Elderly people often maintain homeostasis with a very narrow margin of safety. They seem to enjoy reasonably good health by combining a diet deficient in calories, protein, vitamins, and water with a protected and clocklike life of restricted activity. Because of this fact, careful study of each patient is imperative, with particular reference to the body systems known to degenerate with age. Such study will afford an index of the physiologic age and the reserves of the various systems. Investigation of the functional status of the heart and blood vessels, the lungs, kidneys, and liver, should be done. Routine laboratory examinations should include, beside blood counts and urinalyses, hematocrit, blood urea and blood chloride, and plasma protein determinations. Were blood volume determinations more widely available, further valuable information would be obtained which might well extend the limits of surgery in the elderly patient, as well as eliminate many fallacies arising from misinterpretation of solute levels and concentrations.

Tests of function should have as their goal the determination of margins of safety under stress, rather than performance at rest. For example, renal concentration and glucose tolerance tests, as well as effort tests of the heart, afford a more valid basis for estimation of the physiologic age than tests performed while the patient is at rest or fasting. Further developments in this field of stress testing will help in selecting the elderly patients who may safely undergo operation. (See Chapter 6.)

Evaluation of the elderly patient's mental and emotional state before operation, and alleviation of existing fears, anxieties, and misapprehensions will be well worth the surgeon's time and effort, and are often the fundamental factors in the patient's decision to consent to operation. Evasive answers will breed suspicion and destroy the confidence gained by candor and honesty. The old are usually wiser and more philosophic than the young and middle-aged. They may be cranky and cantankerous, but they are entitled to the peace of mind derived from learning what is planned, what to anticipate, and what is expected from them in the way of cooperation. It is futile to expect patients to abandon lifelong habits but many of them are sufficiently susceptible to suggestion to make such changes as will be beneficial. Elderly patients

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TECHNICAL CONSIDERATIONS

The elderly are particularly intolerant of trauma. Healing and regenerative processes slow down with the passage of years, avitaminosis, circulatory impairment, hypoproteinemia, and anoxia further retard healing. The lack of

resiliency of the vascular system in the aged prevents rapid adjustment to changes in blood pressure or blood volume. Sudden increases in blood pressure from excessive or over-rapid infusions may produce pulmonary edema. Decreases in blood pressure, especially if sudden or prolonged, give rise to circulatory insufficiency which in turn activates a chain of events with which the vessels cannot cope. Hypotension is prone to set off the fatal train of thrombosis, embolism, infarction, and anoxemia (See p 471). Maintenance of normal blood pressure is most important if these dangers are to be avoided.

Rough, bruising, blood-
gerv

the tissues should
on the operating table should be as relaxed
as possible. Extremes of positioning are to be avoided since they impede the return circulation and increase peripheral vascular stasis. Extreme positions and unduly rigorous retraction are responsible for bruising and muscular strains which aggravate the ordinary aches and pains of convalescence. Satisfactory exposure is not obtained by strong arm methods but by properly placed incisions and competently managed anesthesia.

Choice of the correct incision will obviate extreme positioning, minimize the amount of retraction, and shorten the operation. While muscle splitting incisions may seem theoretically ideal, they frequently afford inadequate exposure and invite contusive retraction. Firm healing occurs after transection of abdominal muscles, provided the enveloping fasciae are properly reapposed. Fear of possible insecurity in the healing of a transverse incision should not obscure its positive advantages in obtaining adequate, relaxed exposure. In practice, transverse wounds are more comfortable during convalescence than wounds of other types, and this is particularly noticeable when the patient gets out of bed on the first or second day after operation.

The selection of fine needles and suture material of small caliber is desirable. Since all sutures and ligatures are essentially foreign bodies, the less material buried the better. Suture material of greater tensile strength than the tissues through which it passes cannot be justified. There will be less tissue reaction if more sutures of fine caliber are used in place of a few relatively coarse ones. Interrupted sutures, while more time consuming are less likely to occlude the circulation of wound or tissue margins than continuous sutures. In the event of infection, one or more interrupted sutures may be removed for drainage without destroying the strength of the entire suture line.

Mass ligation is condemned not only because of its insecurity, but also because it produces an avascular island of tissue which may become necrotic and serve as a culture medium. Careful hemostasis must be observed to prevent puddling of blood or serum and the formation of dead spaces. Neither sutures nor ligatures should be tied so tight as to cut through or produce necrosis of the tissues they penetrate. Secure closure of the peritoneum can be effected only with the patient relaxed. This entails well managed anesthesia and careful approximation of the peritoneal and fascial layers. Such a closure will eliminate the protrusion of a tongue of omentum into the peritoneal suture line, often the first step in wound disruption. All dead spaces should be eradicated by coapting sutures. Since the elderly patient heals slowly, sutures usually should not be removed before the tenth day. So far as pos

sible, all factors should be eliminated that might further diminish tolerance to injury and retard the sluggish rate of healing

MEDICATION AND ANESTHESIA

The aged have a low tolerance for some drugs, for others, their response may be the reverse of the usual pharmacologic action. Barbiturates often excite them. Preoperative sedation is likely to depress the circulation and respiration, and is too often needlessly employed at an added risk to the patient. If sedatives must be given, it should be kept in mind that the elderly require only from one-third to one-half the usual adult dose. The lower metabolism of old age carries with it diminished ability to metabolize fixed drugs such as morphine, avertin, and the barbiturates.

Since the risks of surgery in the aged are admittedly somewhat greater than in younger patients, the selection of the anesthetic agent plays a relatively more important role. (See Chapter 9.) The elderly, generally speaking, require less of any anesthetic agent than a younger person. However, *regardless of the agent employed, an adequate supply of oxygen remains essential.* With the tools accessible to the trained anesthetist of today, every desirable factor in anesthesia should obtain so far as the aged patient is concerned. Even assuming some latitude in the choice of anesthetic, it is apparent that the management of the anesthesia remains the most important factor.

RECOGNITION AND CORRECTION OF DEFICITS

In evaluating the nutritional status of elderly patients, many factors that have bearing on their ability to withstand major surgical procedures must be considered. The surgical welfare of the patient depends on a reasonably accurate estimate of various deficits and their correction as far as possible before operation. The repair of those deficits which are associated with weight loss, anemia, and avitaminosis is most important. While in theory the elimination of all deficiencies before operation would be ideal, in practice many situations demand compromise.

In emergencies, such as intestinal obstruction, the immediate relief of the obstruction is obviously more important than the correction of anhydremia and anemia, which can be combated during and after operation. Malignant lesions, which are often ulcerated and infected, seem to have a prior claim on available new protein whether in the form of food, blood, plasma, or hydrolysate. Obviously the speedy correction of anemia by transfusion should be followed by immediate surgical excision of the growth which is primarily responsible for the anemia and protein depletion. Protein, calories, and vitamins should be added as quickly as possible, but the postponement of surgery in the hope of further improving such a patient's condition would be futile as long as the neoplasm remains. Protein deficiencies may be repaired to some extent while transfusions are being given.

Elderly patients are frequently undernourished because of the wasting of chronic disease or as a result of scanty, unbalanced, finicky diets so commonly chosen by the aged. Good results and satisfactory healing are not possible in the partially starved patient. In spite of apparent homeostasis, protein depletion are often present, increasing age, the tendency to eat less physical activity, but it may reach the

point of semistarvation which will lead to depletion of tissue and blood protein. Even in the absence of disease, an inadequate protein intake results in an insidious wasting of body protein. In contrast to water and electrolyte needs, chronic protein deficiencies are cumulative and may exist over rather long periods. That such deficits are seemingly well tolerated may account for their being so frequently overlooked.

Weight loss follows all operations and unfortunately it has either been regarded as inevitable or its implications have gone unnoticed. Such weight loss means increased destruction of tissue, loss of fluid, plasma, and blood, and is the result of operative trauma, hemorrhage, exudates, and tissue breakdown occurring in infections. An inadequate diet, whether due to anorexia, diarrhea, vomiting, external fistula, or simply insufficient consumption of food and fluid, is paralleled by exhaustion of protein from plasma, liver, and

emptying of the stomach or bowel. Protein starvation dulls the appetite and the resulting anorexia enhances the vicious circle unless it is interrupted by parenteral feeding of adequate amounts of protein.

Anemia. Anemia, by restricted definition a deficiency in hemoglobin, is a sign of protein depletion. It is often a late sign because the hemoglobin deficit may be masked by the reduction in plasma volume. Plasma protein is diminished before hemoglobin, possibly because there is only one fifth as much plasma protein as hemoglobin, which in addition is locked within the red cells. Conversely, hemoglobin will be restored first under any regime of protein replacement before tissue or plasma protein deficits are repaired. One might infer that this preferential synthesis of new hemoglobin was evidence of response to the greater need of the body. Cancer and chronic infection predispose to anemia by some defect in the formation of new hemoglobin as well as by bleeding. Anemic patients tolerate surgery poorly and are vulnerable to shock; their wounds heal slowly. Obviously the first step in the repair of an overall protein deficiency is the correction of anemia by whole blood transfusion. Other methods will be slow since the new protein will fabricate hemoglobin before building tissue or plasma protein, and futile because such methods cannot keep pace with the hemoglobin destruction of progressive neoplastic disease.

Weight loss almost always signifies a reduction in blood volume, which in turn indicates not only a plasma protein depletion but also a reduction in total circulating hemoglobin and red cell mass. The paradox of normal plasma protein and hemoglobin concentrations in the emaciated, anemic patient depends on a reduced plasma volume, which will also give a misleading hematocrit. In cases of cancer, chronic infection, or malnutrition, blood volume is reduced 25 to 30 per cent below normal for weight in health and the reduction in total circulating hemoglobin and red cell mass is even greater. Correcting the hemoglobin or red cell deficits by transfusion, blood volume is restored to normal. Unfortunately, accurate calculation of these deficits entails plasma volume determinations, as well as hemoglobin and hematocrit measurements. They may then be compared with values based on previous weight in health and, using the deficit of either red cell mass or hemoglobin as an index, the amount of whole blood necessary to restore normal blood

fluid components of the body amount to less than $3\frac{1}{2}$ liters, the remainder being interstitial fluid. Serious water deficits can be corrected in a few days by the introduction of fluid orally or parenterally. Generally 3 liters (40 cc. per kilogram of body weight) will be sufficient unless there are abnormal losses from vomiting, sweating, or an indwelling gastric catheter. Urinary output of 1000 cc. or more in the absence of unusual losses usually indicates fluid balance. Lack of thirst and low urinary specific gravity give additional evidence of adequate hydration.

Electrolyte depletion may manifest itself by reduced urinary output and is often associated with dehydration. The kidneys stop secreting salt in an effort to conserve it. If renal function is impaired, salt is retained in the interstitial spaces, provoking edema by fixing water within the body. This tendency is aggravated in hypoproteinemia. In the event of impaired renal function, electrolytes should be replaced, not by salt solution, but as sodium lactate or Ringer's solution, either of which will correct acidosis without intervention of the kidneys. When hypoproteinemia and anemia are present, their correction by transfusion or amino acid infusion will accelerate reestablishment of kidney function.

Normally 5 to 10 gm. of sodium chloride are excreted daily in the urine. To replace a decrease of 100 mg. per 100 cc. of plasma chloride requires 0.5 gm. of salt per kilogram of body weight, e.g., for a patient weighing 70 kg. who showed a drop of 200 mg. per cent, 70 gm. of salt would be required which would amount to 7 liters of isotonic saline solution. Large infusions of salt solution should be postponed until renal function is normal. Unless this caution is observed, such infusions when superimposed on the postoperative protein loss may precipitate edema. A low plasma chloride is not particularly significant in a bed patient. Fluid and electrolyte balance cannot be maintained with diminished plasma protein. Clinical improvement, correction of hemoconcentration, and secretion of adequate amounts of urine are bedside evidence of response to therapy.

Proteins. Sixteen hundred calories is the daily requirement of the average sized patient at bed rest. Without food, he will obtain these first from the hepatic glycogen which is soon depleted, and then from the fat and protein tissues of the body. During complete starvation 80 per cent of the energy requirements will be supplied by endogenous fat. However, the body can be maintained in nitrogen balance if the exogenous source of calories, though restricted to 20 per cent, is composed of a sufficiently large proportion of protein.

The difference in urinary nitrogen excretion under conditions of total starvation (15 gm. of nitrogen per day) and protein starvation (5 gm. of nitrogen per day) amounts to 10 gm. of nitrogen, which represents the end product of protein destruction necessary to supply glucose for energy requirements in total fasting. It is the residue of 62.5 gm. of protein. It is fortunate that under extreme conditions only one-sixth of the total energy requirement need be supplied by the protein tissues of the body. Thus if tissue fat can supply as much as three-quarters of the caloric requirement, it is simple enough to make up this deficit with glucose. One hundred grams of glucose a day with the utilization of endogenous fat will meet the body's caloric requirements without using protein for calories.

Muscle tissue contains 20 per cent by weight of protein but will not

serve as a reservoir for protein even if the energy requirements are met, because plasma and liver as well as muscle proteins become depleted on an inadequate protein intake. Although the daily requirement under normal conditions is about 1 gm of protein for each kilogram of body weight (*i.e.*, 70 to 100 gm of protein daily), under conditions of trauma, burns, infection, fever, and after operation the breakdown of protein tissue is excessive and the daily requirements may be two or three times as great to keep the body in nitrogen balance. Acute hypoproteinemia, as well as anemia, occurs with any extensive blood loss. Surgical shock due to hemorrhage is the result of loss of plasma and not loss of red blood cells. Plasmapheresis experiments have shown that shock followed the loss of plasma alone, if protein concentration were sufficiently reduced. Death from severe hemorrhage is due to the inability of the body to replace the loss rapidly enough with fluid containing sufficient protein, and indicates the need for meeting the deficit with whole blood or plasma. It is regrettable that up to now there is no known means of accelerating replacement of endogenous plasma protein from muscle tissue. Protein losses are cumulative because, once lost, protein cannot be replaced by any other food element, even when available sources of carbohydrate and fat spare protein as a source of energy.

During starvation, a patient may lose as much as 100 gm of protein a day over a period of time, which when converted to muscle protein amounts to 500 gm, or roughly four times the ordinary daily physiologic loss. In debilitating diseases when weight loss occurs the net loss in dry protein amounts to about one fifteenth the loss in body weight. Another simple method of approximating total protein loss in grams consists in multiplying the grams per cent decrease in plasma albumin by 1000. The rapid replacement of gross protein deficits constitutes a difficult problem. Protein intake should be as large as possible but need not repair protein deficiencies completely (See also Chapter 12).

The practical methods of protein administration are oral and parenteral. The ingestion of 100 gm or more of protein may not be possible, especially in the presence of anorexia or some other factor limiting oral feeding. One hundred grams of protein may be visualized as the amount contained in four fillets of beef, three and a half quarts of milk, or seventeen eggs. Either plasma or amino acid mixtures may be introduced parenterally to correct protein deficits.

Plasma supplies protein directly to the blood stream and is valuable in severe cases of hypoproteinemia where *immediate* replacement is necessary. One liter of plasma contains 60 gm of protein, double the amount in whole blood, 9 gm of salt, and also 5 gm of sodium citrate which, unfortunately, is often toxic. Another drawback of plasma is its expense, 1 liter requiring four donors as well as processing. However, it will replenish protein deficits, correct hemoconcentration, and maintain nitrogen balance.

Since proteins are absorbed into the body as amino acids, administration of amino acid mixtures is a physiologic short-cut, eliminating activity of the gastrointestinal tract. These amino acid mixtures are inexpensive and may be injected along with glucose and saline solutions. Amino acids are usually

hydrolysates of casein and pork pancreas contain amino acids and small peptides in the proportion of 70 to 30. These hydrolysates are prepared in a 5 per cent solution, usually with 5 per cent glucose, and the pH is adjusted to 6.5. The injected amino acids disappear rapidly from the blood, indicating the avid appetite of the tissues. Large amounts of water must be administered with the amino acids to permit their synthesis into protoplasm, and glucose should be added to aid in their utilization and spare them for tissue building. Such solutions will maintain nitrogen balance, promote growth, and effect regeneration of serum protein. A liter of the solution as usually administered contains 50 gm. of amino acids, 50 gm. of glucose, and 5 gm. of salt. For modest protein deficits 3 liters are given daily, one containing 50 gm. of amino acids and 50 gm. of glucose, and the other two each containing 50 gm. of glucose, a total of 50 gm. of protein and 150 gm. of glucose, or 800 calories in all. In case of greater protein depletion, 50 gm. of amino acids may be added to one or both of the glucose solutions.

Protein deficits should be repaired as completely and rapidly as possible *before* operation. Complete replacement may not be necessary since the improvement in the patient's nutritional status will be considerable if even one-fourth to one-half of the deficit is repaired. Protein replenishment is warranty against edema in its various manifestations, impaired liver function, and lowered resistance to infection. Much of the edema of the intestinal tract found in longstanding gastrointestinal disease can be corrected by rest, decompression, and protein replacement. If protein deficits are eliminated before operation, the intestinal tract will appear collapsed and healthy, rather than swollen, distended and edematous, and operative stomata will remain patent and function normally from the start, instead of being obstructed wholly or in part by edema—the result of unrecognized hypoproteinemia.

If the patient can eat, he should be encouraged to do so. If vomiting occurs or the surgeon wishes to keep the gastrointestinal tract at rest, parenteral feeding should be employed and need not delay operation, since it may be carried on simultaneously with routine preoperative measures.

After operation, fluids such as tea, broth, fruit juices, or carbonated drinks are given by mouth as soon as the postanesthetic nausea subsides. A simple high protein drink consists of 100 gm. of skimmed milk powder in a glass of milk. A liter of this mixture provides over 1000 calories and 100 gm. of protein. Numerous commercial protein hydrolysates, fortified with vitamins, minerals, and carbohydrate, are now available for oral feeding. They are all fairly expensive (\$5 to \$6 a pound). They smell worse than they taste but can be made palatable by mixing in chilled vegetable juices, or by making a thick paste and then diluting it with chocolate milk. Some patients prefer to take them dry and wash them down with water or milk. In the event of suspected vitamin depletion it is safer to give adequate doses of the deficient vitamins than to depend on the vitamin content of proprietary foods.

The correction of protein deficiencies stimulates the appetite and the addition of some fat to the diet aids in the satisfaction of the sense of hunger. The aim should be to put the patient on a normal diet as soon as possible. Once the elderly patient is back on a satisfactory diet due consideration should be given to his whims and idiosyncrasies as far as the potentialities of the diet kitchen permit. The digestive capacities of the aged are probably better conditioned to their individual likes and dislikes than to dietary rules.

Hamburger or corned beef and cabbage may make far greater appeal to the digestive enzymes of the elderly patient who craves such food than would unwanted servings of junket, cereal, or gruel

Weight loss can be minimized and tissue destruction combated by deliberate replacement of known, suspected, and anticipated losses of protein so that healing and repair will immediately follow the injury of operation. When both anemia and hypoproteinemia are present, the combined use of blood transfusions and infusions of amino acids is indicated, the former to repair deficits of hemoglobin and red blood cells quickly, and the latter to supply new plasma protein which will also aid in the synthesis of new hemoglobin (hematin and globin)

CONVALESCENCE AND COMPLICATIONS

There is nothing novel about the concept of early mobility and ambulation. It has long been known that prolonged conventional "bed rest" was illusory following operations on children and the insane, yet their wounds healed readily and their convalescence seemed remarkably easy. In geriatric surgery, the textbook dictum of ten to fourteen days in bed after operation is not acceptable because of the risk of pulmonary and vascular complications. The validity of the practice of moving elderly patients frequently and getting them on their feet early more than justifies adoption of the same routine for the young and middle-aged. Since planned mobility and uncontrollable activity in the young have produced easy convalescences free of complications, with rapid, secure wound healing, the same principle is now being applied to patients of all ages with equal success. Abandoning the tradition of relying solely on nature during convalescence, an active program aimed at satisfying the physical and psychologic needs of the patient has shortened convalescence and reduced complications to a minimum.

Early mobility has many advantages for the elderly patient. It reduces the incidence of postoperative pulmonary and vascular complications. Lowered vital capacity returns to normal much more rapidly when the patient moves about, preferably out of bed. The probability of pulmonary complications falls in proportion to the rapidity of the return of normal vital capacity, which accelerates pulmonary circulation and augments oxygenation. Atelectasis is caused by the accumulation of viscid bronchial secretions in the basal pulmonary segments and a high splinted diaphragm, the latter resulting from the unfortunate combination of the supine position and reflex fixation of the abdominal muscles. It is relieved by increased pulmonary inflation and voluntary coughing. Oversedation dulls the cough reflex and depresses respiration. Pulmonary complications aggravate protein depletion by further loss of plasma into the lungs.

Getting the patient out of bed on the first or second day after operation will largely eliminate increased peripheral venous stasis which is primarily responsible for thrombosis and embolism. Hemoconcentration and increased blood viscosity do not play significant roles since their presence should be decreased by the presence of additional fluid by the oral intake of fluids. The patient's activity decreases circulation time and puts pressure on the calves, collapsing their

veins, in fact, it is the common etiologic factor in pulmonary emboli regardless of the underlying disease.

Reluctance to insist on early activity and early ambulation has been based on suspected insecurity of the wound, due to lack of confidence in the tensile strength of suture material. Even when catgut is employed it has been shown that wounds are stronger with early mobilization than when the patient is kept quiet and in bed. Experimental and clinical studies have shown that early activity accelerates wound healing by hastening fibroplasia and curtailing the "lag period," and that postoperative hernias and wound disruption occur less frequently than under the conventional regime of bed rest. Mobility increases the supply of blood and oxygen, improves the nutrition of tissues, and increases the elimination of metabolites. The correction of anemia, hypoproteinemia, and avitaminosis will cancel out the chief factors in delayed wound healing. Infection, persistent cough or vomiting, distention, especially in the undernourished elderly patient, are the exciting causes of wound disruption and should be combated by appropriate measures. Nonabsorbable sutures will minimize the possibility of postoperative hernias and wound dehiscence. Interrupted sutures of fine cotton, silk, or stainless steel wire in the closure of fascial layers, and the elimination of dead spaces by coaptation sutures, warrant complete confidence in the security of wounds.

There is nothing more enervating than prolonged rest in bed. Muscular weakness, especially when superimposed on malnutrition, will quickly follow disuse, setting in motion a vicious train of circumstances: increasing weakness, loss of muscular tone, and further lagging of the circulation, which establish a perfect field for the formation and propagation of thrombi. Lowered metabolic rate and increased disinclination to eat are followed by complete apathy, asthenia, and inanition. Decubitus ulcer may be considered an index of the depth to which vitality has fallen, and only too often indicates that the regrettable process is irreversible.

In contrast to this gloomy picture, early ambulation relieves boredom, increases interest, and improves the morale of the patient, as well as his muscular tone. It minimizes "deconditioning," loss of muscular abilities, and the atrophies of disuse. It makes for a happier, more cheerful and cooperative patient, provided of course that he understands the rationale, purpose, and safety of the routine. "Keep moving please" should be the motto for every elderly patient after operation. There is less nausea and vomiting and abdominal distention. Prompt return of normal bowel and bladder function usually follows escape from the bedpan, corroborating the observation that the recumbent position is unsuited for functions of elimination. Early activity improves appetite and the increased intake of food augments the strength of the patient, inciting him to more activity which in turn further stimulates his appetite. This sequence of events is one of the great benefits of early ambulation.

Bed rest can be justified only as long as some concrete benefit derives from it. The contraindications to early ambulation are general bacterial peritonitis, excessive weakness, serious hemorrhage at or soon after operation, thyroid crisis, recent coronary occlusion or thrombosis, and pulmonary embolism. Fever, pulmonary complications excepting infarction due to embolism, intraperitoneal abscess, distention, draining wounds, and tubes in hollow viscera are not absolute contraindications. No patient should ever be forced to sit up or get out of bed. However, if he understands the advantages

which will obtain from early postoperative mobility, he will usually cooperate, particularly if he is in a small ward where he can observe the obvious physical and emotional benefits which other patients gain from activity

The chief complications of the elderly patient involve the respiratory and vascular systems. There is a relative absence of severe systemic manifestations and reactions in the aged. In the absence of complications, they are less sick, less prostrated, exhibit less fever and relative weakness than younger patients undergoing similar operations. To be sure this postoperative pattern carries with it the danger that complications may be masked or overlooked, and certainly they are more dangerous in the aged than in younger patients. When complications do occur in the elderly patient, they must be combated vigorously and without delay, since in twenty-four hours too much ground may be lost ever to be regained. The responsibility for postoperative supervision and care of the elderly patient should not be delegated to anyone unfamiliar with the particular problems of the aged

SUMMARY

With a constantly increasing percentage of the population reaching ages over sixty-five, growing numbers of elderly people need surgery. Cancer occurs more frequently in the elderly, and age does not preclude the other common ailments amenable to surgical treatment. Thus the surgeon needs to familiarize himself with the special problems of geriatric surgery.

The aged are handicapped by retarded healing and regenerative capacities, reduced margins of safety in the various systems, and numerous nutritional deficiencies.

Competent management of anesthesia, which must provide a constant and adequate supply of oxygen and effective resuscitation technics, is indispensable. Replacement of blood and fluid as they are lost during operation to stabilize blood pressure is an additional function of the anesthetist.

A careful preoperative assay of the elderly patient will point to the particular deficiencies in the nutritional status which should be corrected as completely and rapidly as possible *before* operation. Most important is the repair of deficits in protein and blood, manifested by weight loss and anemia. Deficiencies in fluid, electrolytes, calories, and vitamins should also be restored, before, during, or shortly after operation.

Early postoperative mobility and ambulation eliminate the enervation and hazards of prolonged bed rest and shorten convalescence. The patient's *understanding of the rationale and purpose of this regime is essential* to its success.

The ability of the aged, when properly prepared and cared for, to with-

for many necessary surgical procedures

CHAPTER 9

PRINCIPLES OF GERIATRIC ANESTHESIA

RALPH T. KNIGHT

To the anesthetist old people present certain characteristics which differ from those of the average subject for anesthesia

GENERAL CONSIDERATIONS

Mental Factors The mental attitude of old people is, on the whole, more receptive. They have passed through a period of adult prime in which self discipline, control, and their best efficiency have been achieved. They have retained the discipline in large degree and have added to it more patience and less concern and fear of new and unknown experiences and dangers. As age advances, a larger proportion of their friends have had surgical experiences and these have become accepted as hurdles to be more or less expected and taken in their stride. Moreover, as age advances, more and more of life's responsibilities have been fulfilled, goals attained or, perhaps, lost. Whatever may eventuate becomes more acceptable in contemplation. In fact, even the contemplation of eventualities becomes a less frequent occupation.

In general medicine this attitude is probably a disadvantage, for it lessens the "fighting spirit" which is the physician's ally. In anesthesia, on the other hand, the peaceful and unconcerned approach is a decided advantage, equally for induction, smooth maintenance, and recovery. Many older and aged people combine a happy will to live with a simple preparedness for death. This makes them especially favorable subjects for anesthesia. In my experience old people are especially easy to meet before anesthesia, and very responsive to a little sympathetic bantering.

Physical Factors Such preparations as positioning on the table are very difficult for most oldsters. It is hard for them to understand what is wanted. Above all, they cannot make their bodies relax and let others move them as desired. Arms, legs, back, and neck are apt to be held rigidly in a confused attempt to do as told. Requests that the muscles be relaxed, loosened, and let go often even increase the tension. Then too joints and muscles are often painful when moved. One must have utmost patience, persistence, and care with old people during the rather electric time in the operating room, when all present are anxious to proceed. When general anesthesia is to be employed, it should usually be induced before manipulation or positioning is attempted. It is especially important to use enough pillows under the head, neck, back, and knees, or wherever indicated by the conformation of the body.

In old people, reflexes are much more easily subdued than in younger subjects and decreasing muscle tone makes complete relaxation more easily achieved.

PREANESTHETIC MEDICATION

Preanesthetic medication is an important consideration. Certainly, for patients of all types, adequate sedation makes for greater facility, smoothness,

efficiency, and safety in all kinds of anesthesia. Many physicians have an inordinate fear of administering sedatives, especially morphine, to elderly people. Others make the mistake of ordering routine adult doses without special consideration of the patient's debilitated condition. The state of the subject's vigor is the important criterion for the dosage. The metabolic rate, that is the total oxygen consumption, is probably what we are trying to express when we speak of vigor. If we could measure the oxygen consumption just before giving the sedative, we should arrive at the suitable dose very accurately. Practically, we must depend upon a considered evaluation of a combination of such factors as age, size, occupation, habitual activity, recent activity, muscle tone, hemoglobin, appetite, recent loss of weight, confinement, pain, and the recent necessary use of sedatives and analgesics.

Morphine and Hyoscine. According to one's appraisal of these factors in the individual case, one should administer somewhere between $\frac{1}{4}$ grain of morphine with $\frac{1}{150}$ grain of hyoscine and one-sixth of those amounts, *i.e.*, $\frac{1}{24}$ grain of morphine with $\frac{1}{800}$ grain of hyoscine. Atropine may be used instead of the hyoscine in somewhat larger amounts, but the hyoscine does very well in adding to the mental quietness in lieu of any barbiturate. The hypodermic should be given forty-five to sixty minutes before the induction of anesthesia and I have found no reason to vary the dose preceding different types of anesthetics. In the geriatric group of patients, the dose of morphine should seldom exceed $\frac{1}{4}$ grain. It usually ranges between $\frac{1}{8}$ and $\frac{1}{16}$ grain. On the other hand, it should seldom be omitted. The accompanying dose of hyoscine should be approximately $\frac{1}{32}$ as much as the morphine. If atropine is used instead of hyoscine, it is well to give between $\frac{1}{4}$ grain and $1\frac{1}{2}$ grains of pentobarbital sodium an hour before anesthesia, and as careful consideration should be given to this as is given to the dose of morphine. Old people take sedatives very well according to this plan.

No other drugs seem to be improvements over these mentioned. Recently there has seemed to be a revival of agitation against the belladonna drugs on the ground that they cause the mucus to be thicker and more tenacious, which may encourage atelectasis. On the other hand, without one of these drugs, there is apt to be a very copious flow of saliva and mucus, often seriously flooding the tracheobronchial tree and interfering with oxygenation and even with the delivery of an inhalation anesthetic. The presence of secretion about the epiglottis and glottis often causes troublesome and even serious laryngospasm. Some anesthetics do not, in themselves, stimulate secretion. However, in any type of anesthesia, if, because of anatomic and mechanical difficulty in keeping the airway through the nose and mouth well open, it becomes necessary to use a nasal or oral pharyngeal tube, secretion is stimulated and may itself cause laryngeal spasm. The belladonna drugs help to prevent spasm by subduing the parasympathetics. These contending arguments make the question of the belladonna drugs still controversial.

Reaction to Preanesthetic Medication. Occasionally an individual will exhibit a severe reaction to these drugs, with blotchy or confluent flushing, peroral pallor, extremely dry mucous membranes, dry, hot skin, elevated body temperature, and rapid pulse. The elevated temperature and rapid pulse are the most important of these signs and indicate postponement of the operation. The flushed, warm skin is usually the first sign drawn to our attention. One decides according to the severity of the signs. When they are

appearing, however, one should not start anesthesia earlier than an hour after the hypodermic has been given, in order to witness the maximum reaction before anesthesia is induced. Geriatric patients, I believe, are less apt to have adverse belladonna reactions than other adults and especially less than children.

TYPE OF ANESTHESIA

Probably a majority of readers will consult this chapter for rules for indications and contraindications drawn up especially for anesthetizing old people. If so, the text is apt to be disappointing. In a rather wide experience with anesthesia in old people, I have been more and more impressed with the evidence that any anesthetic or method, or combination of anesthetics and methods, that is serviceable and good at all is serviceable and good for old people. The main difference is that these patients are tender, they are easily quieted with sedatives, easily anesthetized and relaxed, and readily overdosed. What they require particularly is conservatism with a little daring mixed in, but with unceasing vigilance and nicety and exactness of administration and control.

Intravenous Anesthesia This type of anesthesia has proved itself a boon to old people. Strange contraptions are apt to be appalling to them and they are frequently subject to claustrophobia. Hence a mask is more disquieting than the prick of a needle which they need not see. The oncoming of unconsciousness is rapid and pleasant. *Pentothal sodium* is a drug of choice. It is a little more potent than evipal soluble and therefore can be administered more gently. It subdues reflexes more easily and is accompanied by a much smaller incidence of sneezing, coughing and hiccoughing.

Pentothal Sodium Pentothal sodium should be used in no stronger concentration than 2½ per cent. For old people, after the first 2 or 3 cc. have been injected, there should be a pause of thirty seconds to witness the result. Some will go soundly to sleep and would have been overdosed had the administration continued, even slowly. Some will remain wide awake, which will indicate that the injection of 1 cc. every five seconds until unconsciousness arrives will not be too much. The patient should then be kept as lightly anesthetized as possible. Slight reflex movements should even be allowed and increase in reflex motions should constitute the principal sign that more anesthetic is required.

To insure a rapid awakening, one should always plan the administration, if possible, so that no additional pentothal need be given during the last ten minutes of any procedure. Any amount given during that time lengthens the sleep out of all proportion to the added dose. Because respiration tends always to be depressed by pentothal, and old people are sensitive to oxygen deficit, one should administer oxygen habitually, except during very short procedures.

INDICATIONS AND CONTRAINDICATIONS It seems to me that pentothal sodium anesthesia alone should not be used, especially for old people, in operations requiring much relaxation. Their brains are easily anesthetized, and after barbiturate anesthesia for such operations, they remain asleep too long a time. It is the ideal anesthesia for biopsies, operations on the lower urinary tract, which can be done in forty-five minutes or less, and for reduction of fractures. Good relaxation for the latter purpose can be easily obtained at the end of induction. One should not attempt to prolong relaxation, however.

because when blood stream and tissues have all become well loaded with the anesthetic, one is close to an overdose, and if that occurs under these circumstances it is hard to combat. In addition to the operations named above, innumerable other occasions for intravenous anesthesia suggest themselves. For instance, it is suitable for the amputation of a gangrenous extremity, in diabetics or in those with peripheral arterial disease. It does tend to raise the blood sugar but this is temporary and is not believed to cause any ill effect, even in diabetes.

Contraindications to pentothal are few. The most definite one is cardiac decompensation. The concurrent use of the sulfonamides as a contraindication is practically obsolete. Liver damage must be gross to be important.

Curare is an intravenous agent introduced into anesthesia in 1942. Its present most satisfactory preparation is solution of tubocurarine, 3 milligrams (20 units) per cubic centimeter. This has no anesthetic action but produces muscular relaxation by its effect upon the neuromuscular junction. It can well be administered with, even actually mixed with, pentothal sodium. An excellent proportion is 15 mg (100 units) of tubocurarine to 500 mg of pentothal sodium. By use of this combination the patient is kept quiet although very lightly anesthetized, the amount of pentothal administered is astonishingly reduced, and the sleep therefore greatly shortened. Relaxation may be easily obtained for any surgical procedure.

Because of its convenience, one is tempted to use intravenous anesthesia for biopsies from the oral cavity and tongue and for the insertion of radon in that region. This is permissible if every precaution is taken to prevent respiratory obstruction by muscular spasm, mechanical displacement of the tongue, and the like, and the entrance of blood and debris into the trachea. These things are more apt to occur with pentothal anesthesia than, for example, under ether. In most such instances a tracheal tube should be inserted before the surgical procedure is started. This insures control of the situation under any circumstances and the prevention of nearly all complications. Tragedies occur when the whole procedure is taken too lightly and one is caught unprepared.

Spinal Anesthesia. In geriatric surgery there is a very useful place for spinal anesthesia. Any of the drugs to which one is accustomed can be used well for old people. The chief difficulty is the relatively high incidence of inelasticity in the circulatory system so that it does not accommodate well to physiologic upsets which tend to cause changes in blood pressure. Furthermore, old people's tissues, notably the heart muscle and brain, are especially susceptible to damage by the anoxia and the risk of thrombosis attendant upon acute hypotension, and their small vessels are particularly susceptible to the strain of hypertension. One must exercise marked care, therefore, to keep the blood pressure as near as possible at its preanesthetic level. If the

anesthetist should then inject intramuscularly a dose of the pressor drug which he believes will be sufficient to prevent a significant blood pressure fall, and

before completing the spinal anesthetic procedure are
nal

tap I have seen three deaths due to the optimistic injection of ephedrine before attempting spinal puncture, or the injection of too large an amount. The result was a severe and prolonged rise of blood pressure followed by cerebral accident. The prophylactic dose of the pressor drug should therefore be conservative, about 25 mg of ephedrine. One should be prepared to inject a small amount intravenously immediately if the pressure starts to decline. Ten milligrams is enough, one should then wait, with the needle in the vein, for one minute or a little more to see what effect will take place. Additional amounts in units of 5 mg can then be added as indicated over the next two or three minutes until the blood pressure has become stabilized.

These considerations should not interdict the use of spinal anesthesia as compared with general anesthesia, which also has its disadvantages. They merely emphasize the need for vigilance and attention to detail. The nonchalant and inexperienced administration of spinal anesthesia to old people must be frowned upon. In a large proportion of cases, however, properly used, it offers the least possible risk.

To go into details of selection and technique of administration of the various drugs would require more space than is allotted. A few examples may suffice.

For Control of Blood Pressure. A most effective combination, and one of my favorites, is a mixture of neosynephrin and ephedrine in the proportion of 1 mg of the former to 20 mg of the latter. This is especially helpful for old people with whom one must be cautious not to give too much too soon, but must certainly avoid too little too late. The above amount is a good average initial intramuscular dose. Each intravenous unit should be not more than one fourth or one fifth of this amount. Neosynephrin alone, in doses frequently spoken of (3 mg, for example), is dynamite and frequently sends the blood pressure skyrocketing one hundred and fifty points.

Use of Spinal Anesthetics. Too high a concentration of spinal anesthetic drugs is apt to cause permanent damage to the nerve roots or spinal cord. This has been a much more frequent happening than injury by the needle. One should not inject, and there is no occasion to inject, higher concentrations than 5 per cent procaine, 3 per cent metycaine, 0.5 per cent pontocaine or 1:1500 of nupercaine. The most convenient way to use procaine and metycaine is to measure in the syringe, out of the ampule, the amount of the solution to be used, attach it to the spinal needle and aspirate spinal fluid to the desired dilution.

The 1 per cent ampule of pontocaine is used in the same way, or may be diluted with an equal quantity of 5 per cent dextrose to make it heavier than spinal fluid. The "nuphanoid" preparation of pontocaine may be dissolved in the proper quantity of 5 per cent dextrose or spinal fluid to produce 0.5 per cent pontocaine. The 0.5 per cent ampule of nupercaine may be diluted with 5 per cent dextrose solution to make 1:1500 nupercaine slightly heavier than spinal fluid, or an ampule of the 1:1500 lighter solution may be used, according to the need.

Injection of any of these solutions must be no slower than 0.5 cc per second in order to cause immediate mixture and further dilution with spinal fluid. If injection is too slow, the solution remains in a discrete layer, still strong enough to cause nerve damage, and also still separate enough to roll too fast and too far and for too long a time after injection if the table is slanted. Injection as rapidly as 1 cc per second can do no harm.

Special Operative Considerations For an amputation in the lower thigh or leg, 80 to 100 mg of procaine or metycaine in 5 to 3 per cent solution injected between the third and fourth lumbar spines should give about an hour of anesthesia.

For *transurethral resection of the prostate*, 70 to 100 mg of procaine or metycaine in 5 or 3 per cent solution, injected between the third and fourth or fourth and fifth lumbar spines, with the patient sitting up and remaining so for three or four minutes, should give from forty-five to seventy five minutes of anesthesia. The dome of the bladder will remain sensitive to distention. Some surgeons prefer this. This dosage also causes a minimum of blood pressure fall which, if necessary, is well controlled by intravenous fluid. Some surgeons prefer intravenous saline without the use of any pressor drugs because any inadvertent rise of blood pressure causes difficulty in controlling hemorrhage. If it is desirable to relieve the patient of any sense of distention, it is necessary to inject the above amount between the second and third lumbar spines, with the patient in the lateral recumbent position, and to give about 25 mg of ephedrine to prevent hypotension.

For *inguinal herniorrhaphy*, 100 to 140 mg of procaine or metycaine in 5 or 3 per cent solution, injected between the third and fourth lumbar spines, should establish from sixty to ninety minutes of anesthesia up to the tenth or eleventh thoracic segment, as needed for unilateral or bilateral operations.

For *appendectomy*, *suprapubic prostatectomy* or *pelvic laparotomy*, 100 to 140 mg of procaine or metycaine injected between the second and third lumbar spinous processes should establish sixty or ninety minutes of anesthesia up to the ninth or tenth thoracic segment. More than a very slight Trendelenburg position should not be instituted until at least ten minutes after the spinal injection, because these solutions are definitely heavier than spinal fluid and until adequate mixture and dilution have taken place, they will flow downward toward the head. If anesthesia of longer duration is desired, 12 to 16 mg pontocaine in 0.5 per cent solution, diluted with spinal fluid from the 1 per cent ampule, may be used. As this has the same specific gravity as that of spinal fluid, the Trendelenburg position may be instituted almost at once if desired.

For *perineal prostatectomy*, the choice lies with nupercaine. The position is to be a modified combination of the lithotomy and extreme Trendelenburg positions. No preparation for operation can be begun until this position is established. The positioning and preparation are long at best. If a light 1:1500 solution of nupercaine is used, positioning may be instituted at once. A small

drug should be necessary or desirable because of the low anesthesia and head-down position.

For *hemorrhoidectomy* one may well use either of the technics described for transurethral or perineal prostatectomy. If the Kraske position is to be used, the technic described for perineal prostatectomy is especially applicable.

anesthesia up the sixth thoracic segment is required. From 8 to 12 mg. of nupercaine, according to the size and vigor of the patient, in 1:1500 light solution, are injected between the first and second lumbar vertebral spines and the table is tilted slightly with the head end up 5 or 10 degrees, while the preparation proceeds. During this time, the anesthesia level is tested with forceps until it reaches the xyphoid, at which time the Trendelenburg position is instituted. Anesthesia will usually last three hours.

For *gastric resection*, long and high anesthesia is required. The same technic described immediately above for bowel resection may be employed. To insure anesthesia up to the fifth thoracic vertebra, the procedure may be modified as follows. The 1:200 solution of nupercaine is diluted with 5 per cent dextrose to 1:1500. Eight to 12 mg. in this solution are injected between the first and second or between the second and third lumbar vertebral spines. The patient's head is then raised on a high pillow and the table is tilted with its head end down 10 degrees. The solution has been made slightly heavier than spinal fluid (about 1.01) by the dextrose, and flows toward the head but does not pass the thoracic concavity. Thus the upper intercostal nerves and the phrenic nerve are protected from anesthesia.

For *biliary surgery* this same technic may be used. For the usual cholecystectomy, however, 125 to 150 mg. of procaine or metycaine in 5 per cent solution, injected between the first and second lumbar spines, is generally sufficient. With the patient's head on a pillow the head of the table may need to be lowered slightly to insure high enough anesthesia.

For operations upon the *kidneys and upper ureters*, the choice is nupercaine in 1:1500 light solution. Ten or 12 mg. should be injected between the first and second lumbar vertebral spines while the patient is lying with the operative side up. He may then be adjusted immediately into the surgical position. This causes the light solution to be brought effectively into best contact with the roots most needed to be anesthetized and anesthesia lasts long enough for practically any surgical procedure upon the kidney.

Continuous or Fractional Spinal Anesthesia. The development of continuous or fractional spinal anesthesia has been a great help in the treatment of malignancy of the gastrointestinal tract in old people. With the use of the Lemmon mattress, soft needle, and extension tubing, the needle may be left in place and additional doses of 5 per cent procaine may be added as needed to prolong the spinal anesthesia. Pontocaine is also an excellent drug for this purpose, especially if weighted with a little dextrose. Reports by Lemmon, and by Woodbridge, Eversole, Hand, Nicholson *et al.*, from the Lahey Clinic, should be studied for variation in technic.

It has now become an accepted practice to add ephedrine, 25 mg. to 50 mg., to the spinal anesthetic solution. This delays absorption of the drug and prolongs the anesthesia very considerably. It has practically no effect upon the blood pressure. Perhaps its favorite use is with pontocaine frequently prolonging the anesthesia with that drug to three hours.

Adjuvant Medication. The greater the dose of anesthetic, and the higher the reach of anesthesia, the greater is the tendency for the blood pressure to fall and the larger should be the prophylactic dose of a pressor drug, up to 50 mg. of ephedrine or 1.5 mg. of neosynephrin with 30 mg. of ephedrine may be required.

Because old people are especially susceptible to oxygen lack, oxygen

should be administered almost routinely throughout spinal anesthesia. This is especially important when the anesthesia is high.

Refrigeration Anesthesia For amputation of extremities, gangrenous because of vascular disease, refrigeration anesthesia is an excellent choice. A circular ice pack is applied at the proposed location for the tourniquet for about an hour. The limb is then raised, the tourniquet applied, and the limb completely packed in ice to a level above the tourniquet. After three or more hours of continuous refrigeration in this manner the limb may be amputated without sensation, and with little or no effect upon the patient's general condition.

In combination with spinal regional, or refrigeration anesthesia, general anesthesia should be used freely, especially for old people, to relieve and prevent any kind of distress, such as fatigue, nausea, discomfort of unanesthetized parts of the body due to position, etc. Nausea is especially troublesome if the surgical region is the upper abdomen. It almost invariably occurs during such surgery under spinal anesthesia, is very distressing to the patient, and makes the task of the surgeon infinitely more difficult. General anesthesia to the point of unconsciousness only is all that is necessary. One must never feel defeated or judge the spinal anesthesia unsuccessful or useless just because accessory general anesthesia is added. General anesthesia alone for such cases must be very profound and in the writer's opinion adds greatly to the depression from which the patient must recover and prolongs the recovery often by many days even if no complications are evident. Recovery from spinal anesthesia, even with light general anesthesia added, is much more prompt, life seeming to pick up from the immediate preoperative period with much less interruption.

Pentothal is an excellent adjuvant. The gases do very well also, cyclopropane being the best and ethylene next.

Inhalation Anesthesia When an inhalation anesthetic of any kind is to be given to an old person his comfort is greatly increased if unconsciousness is induced with pentothal followed by any other anesthetic selected.

Nitrous Oxide is the poorest of all anesthetics for the aged. The patient's greatest anesthetic danger is anoxia and nitrous oxide keeps him on the verge of it. Its only virtue is that it is not explosive if no ether is added. It will accomplish nothing which a little pentothal will not do and with the latter an abundance of oxygen can always be given. There is one good use to which nitrous oxide can be put in geriatric anesthesia, however. If a mixture of 50 per cent nitrous oxide with oxygen is inhaled during pentothal administration, the amount of pentothal needed is reduced and nonexplosive conditions are maintained while an abundance of oxygen is still provided.

Ethylene is a very useful anesthetic for old people for a wide variety of procedures of a medium and minor nature. It is not, however, very much more potent than nitrous oxide and if one tries to accomplish too much with ethylene and oxygen alone, he frequently finds his patient on the verge of oxygen deficit or even in it. It is much better to add a little of a more potent anesthetic, such as cyclopropane or ether, and to maintain the oxygen at an unmistakably high level. When adding ether, one's tendency is to depend

an adjuvant, is much more of a depressing and debilitating drug than I like to give to old people. It seems to leave them much more fatigued and debilitated during the days following surgical operation than other forms of anesthesia.

Cyclopropane is a most excellent anesthetic. Its potency allows the simultaneous administration of plenty of oxygen. For minor operations it provides very rapid, pleasant induction and rapid pleasant awakening. For major operations, especially if 'controlled' respiration is used, relaxation equal to that obtained with ether can be induced. This is particularly true for old people. If allowed to become too concentrated in the heart blood, by too rapid an increase of cyclopropane in the mixture, it causes extrasystoles in some people, and if this condition is allowed to progress, ventricular tachycardia and even fibrillation may supervene. The heart effect, however, is readily reversible, and develops rather slowly, and, if one is at all alert, the rhythm can be brought back to normal in a few seconds by replacing the mixture in the bag with oxygen and increasing the exchange by intermittent hand pressure upon the bag during inspiration. Cyclopropane also sometimes causes an increase in blood pressure, which usually can be corrected in the same way. If with patience and gradual increase of cyclopropane it is impossible to achieve sufficient relaxation without these untoward effects, it is better to stop short of the desired depth of anesthesia and accomplish the rest with a little added ether.

Cyclopropane is almost, but not quite, an ideal general anesthetic. In my opinion it does much less harm to the patient than ether. However, after

or cyclopropane for this purpose, together with oxygen.

The greatest recent advance in anesthesia—especially for old people, is the combined use of pentothal sodium, curare, and nitrous oxide-oxygen. Anesthesia is induced with pentothal-curare. The mask is then applied with nitrous oxide flowing 500 cc and oxygen 500 cc to 700 cc per minute. An intratracheal tube is inserted if indicated. Small additional amounts of pentothal and curare are added as needed. Anesthesia suitable for any surgery may be thus maintained and the patient awakens in astonishingly good condition.

SPECIAL SAFEGUARDS

Old people's faces are often difficult to fit perfectly with the usual mask. It is helpful to fill the cheeks with gauze to round them out and bring the skin into contact with the rubber rim. An oropharyngeal airway should be inserted as soon as anesthesia has advanced sufficiently for the patient to tolerate it. This guards against obstruction and also rounds out the edentulous face to improve the fit of the mask.

Whenever, under general anesthesia, there is the slightest doubt of ability to maintain an adequately patent airway throughout the entire length of the anesthesia, a tracheal tube should be inserted at the outset.

Any degree of inspiratory obstruction leads easily to pulmonary edema and to anoxia. The use of the tracheal tube allows the removal of mucus

from the tracheobronchial tree by suction catheter passed through the tube and thus helps greatly in the prevention of atelectasis

At the conclusion of every general anesthesia all mucus and saliva should be aspirated from the nose, mouth, and oropharynx whether a tracheal tube has been used or not

Every unconscious patient should be transported from the operating room lying upon his side to prevent the inhalation of vomitus and mucus and the backward gravitation of tongue and jaw. Every anesthetist who conscientiously maintains this position during transportation and until the patient is fully conscious in bed will save several lives during his career

CHAPTER 10

PRINCIPLES OF PHYSICAL THERAPY FOR THE AGED

RICHARD KOVACS

THE principal systemic changes in old age are the wearing out of tissues through vital processes, and the increasing difficulty of anatomic and functional restitution without real pathologic changes. These and other irreversible changes call for conservative methods of treatment such as are offered by physical agents. Physical measures improve function and counteract the progress of disease processes. The benefits of these agents on conditions of old age are instinctively recognized by animals and men: the old dog lies in the sun and takes a sun bath, old people love to sit in the sun, especially on cold days. It is said of King David in the Book of Kings: "Now King David was old and stricken in years, but he got no heat. Wherefore his servants said unto him, 'Let there be sought for my lord the King a young virgin, and let her stand before the king, and let her cherish him, and let her lie in thy bosom, that my lord the King may get heat.'"

The modern development of physical medicine has greatly increased the scope of physical therapeutic measures. Old people, especially in the United States, have been eager to take advantage of these measures. Not only do the privileged classes flock yearly to climatic resorts and to the southern sea shore in an effort to stay comfortable during the winter months, but the great mass of old people suffering from rheumatic and other disabilities have learned to expect relief and restoration of comfort and function from all year round use of physical measures as part of a well planned therapeutic regime. Sollman¹ states, "Although drug therapy and drugless therapy may seem direct antipodes to the superficial thinker, they involve the same principles, evoke the same phenomena, accomplish the same results. They differ only in the means which they employ, of which sometimes the one, sometimes the other is better adapted to secure the desired end. Indeed, the differences between physical therapy and pharmacochemical therapy are no greater than those between radiant and direct heat, or between local and general anesthetics."

In view of the spectacular development of many new types of apparatus for the administration of physical treatment, physical therapy must not be confused with apparatus therapy, for the basic effects of simple forms of heat, light, water, massage, and exercises are essentially the same as those of the more complicated types of applications. Any form of physical energy applied to the body exerts first a physical action known as the "primary" physical effect: thermal, chemical, or mechanical action. The primary physical effect brings on "secondary" physiologic effects and these are the major basis of the subsequent clinical effects. The influence on organic or functional changes in the body will be in direct relation to the primary physical changes, hence the necessity of a careful technic and measured dosage in the application of

all physical agents. As a general rule, by the same token, physical therapy should be toned down in the aged, like drug therapy.

In presenting the principles of physical therapy as they are applicable to the aged, we will endeavor to describe briefly each physical method, its physiologic effects, clinical uses, and the possible contraindications and dangers.

The methods presented include

Thermotherapy	Light therapy
Hydrotherapy	Electrotherapy
Mechanotherapy	

THERMOTHERAPY AND CRYMOTHERAPY

General Considerations of Thermotherapy. Heat is one of the universal forms of energy into which all other forms of energy are convertible. It is also the most valuable and most versatile physical force for treatment. Heat can be defined as the internal vibration of the molecules of which a body is composed. Cold is a negative physical state, depending upon the decrease in the amount of molecular vibration that constitutes heat.

The human skin may be regarded as a great sheath of imperfectly sheltered blood vessels and nerve endings, and the effect of thermal measures essentially a reaction in the skin and in the organs reflexly connected or directly penetrated. It has been shown by physiologists that there are special nerve endings, "receptors," for heat and cold in the skin. The more intense the stimulus, the larger the surface of skin it affects and the longer it lasts, the more intense will be the reaction caused by it.

Heating may be applied to the body by (1) conduction from water bath, compresses, poultices, or electrically heated pads in close contact with the body, (2) by radiation from luminous or nonluminous sources—the most convenient and safe method in the aged—and (3) by conversion of high frequency electrical energy in the form of long and short wave diathermy. In this section heating from radiant sources and fever therapy only will be presented, the other applications of heat and cold will be discussed under Hydrotherapy, while diathermy will be presented in the section on Electrotherapy.

Physical and Physiologic Effects of Heating. No matter what form of heating is applied to the body, its immediate effect is purely physical, a rise of temperature in the tissues. The degree and extent of the primary heat effect will vary according to the source of heating, its intensity, and the length of

chronic inflammatory changes in traumatism and in the various catarrhal
as the gonococcus and requires adequate dosage at 100 / 1 or over, sustained
for hours

General heating serves to increase general metabolism and increase elimination through the skin, lungs and kidneys, it also produces a shift toward systemic alkalosis

Types of Heating. Radiant Heating The sources of radiant heat are metallic conductors which become heated by the passage of an electric current. We speak of low temperature or nonluminous sources, or infra-red generators and high temperature or luminous sources or heat lamps. The local effect of heat lamps or infra-red generators does not differ materially. Within a few minutes after exposure the skin becomes red and feels hot. The redness appears in the form of red spots or a network of red lines, and it persists from ten minutes to one hour depending upon the length of exposure.

The advantages of heat radiation over methods of conductive heating (hot-water bottles, poultices, etc.) are (1) that its action extends to a greater depth, (2) that there is no pressure over the parts, and (3) that the parts may be kept under constant observation without difficulty and thus signs of undue heating can be discovered immediately.

Indications for the local use of radiant heating overlap those from conductive and convective sources. The principal indications for local heating from any source are (1) subacute and chronic traumatic and inflammatory conditions in accessible locations (such as contusions and muscle strains, traumatic synovitis and tenosynovitis, sprains, dislocations, and fractures), (2) acute, subacute, and chronic catarrhal conditions of the mucous membranes in accessible locations (conjunctivitis, coryza, sinusitis, bronchitis), (3) infections of the skin (folliculitis and furunculosis) in which it is often advantageous to combine radiant heat with ultraviolet rays, and (4) various arthritic and rheumatic conditions. As a rule aged people relish mild heat applications especially for the last group of these indications.

The *general application* of heat is indicated in conditions of impaired metabolism, in rheumatoid arthritis, and in certain forms of nephritis. General nervous sensitiveness is usually markedly lessened, although too prolonged and excessive heat applications may cause profound depression. General application of heat is administered by electric cabinet baths, hot air ovens, or by various hydrotherapeutic procedures.

Artificial fever treatment (hyperpyrexia) by baths, heat radiation, or diathermy has found a definite place in therapeutics to increase the protective and defensive mechanism of the body. Mild degrees of fever, from 101° to 104° F., maintained for three to six hours, have been found effective in neurosyphilis, ocular syphilis, and resistant seropositive syphilis, as well as in selected cases of arthritis and rheumatoid disease, including painful neuritic and radicular conditions. Age less than sixty is no contraindication *per se* to fever therapy, if administered by competent medical men. The recognized contraindications are myocardial degeneration or valvular, coronary, or other cardiac abnormalities, impaired renal function from organic disease, excessively high blood pressure or arteriosclerosis, tuberculosis, diabetes, far advanced syphilis of the central nervous system, and chronic alcoholism.

Contraindications and Dangers. As all forms of percutaneous heat treatments are usually administered within comfortable toleration of the patient, special care and clinical experience is needed over anesthetic areas in all persons and especially in old people because of lessened skin sensibility. In patients with diabetes, overheating may cause gangrene, especially of the

extremities. The same danger exists in peripheral vascular disease, where ill-considered local heating procedures have caused irreparable damage. In these cases thermostatically controlled "baking" at 94° F or lower is fairly safe, also high frequency heating of the thighs or the lower abdomen, bringing about an increased arterial supply from above.

In addition to these general contraindications, one must take into consideration the fact that the thermal reactions of old people differ from those of the normal individual in the prime of life. Romberg describes two kinds of abnormal reactions of sclerotic blood vessels. Either they do not respond to a thermal or caloric stimulus by contraction or they respond with a cramp and remain in protracted contraction. These differences must be recognized when applying thermal agents—hydrotherapy, radiant light and heat, and diathermy—to old people. As a rule, it can be said that they stand overheating or chilling equally badly. An extensive thermal application for an arthritic process often leaves the patient quite weak and exhausted. Others, when leaving the physician's office after some not necessarily extensive thermal application, become chilled and report that following treatment they had to take to bed with a cold. It should therefore be an established rule to proceed slowly with both the degree and duration of the application of heat and cold in the aged, avoiding extremes as well as overdoses of any potent measure.

The careful home use of heat lamps or infra red generators is often advisable in old people who cannot come frequently to physician's offices, this enables more frequent applications at low intensity and also avoids the danger of "catching cold" after travelling home following an office treatment.

Ice packs, ice water, or mechanical refrigeration have shown its value in some surgical and traumatic conditions. In gangrenous extremities cold acts as a preoperative anesthetic, in compound fractures, severe burns, and crushing injuries it also enables the control of hemorrhage, infection, pain, and progressive shock. In peripheral vascular disease, as well in "immersion feet" and frostbite, it has been shown that reduction of temperature reduces cellular metabolism, makes the blood supply adequate, and prevents gangrene. Hence in these cases the judicious use of cold has resulted in marked relief of pain, subsidence of edema, and a minimal amount of tissue damage. It is also of definite value in freezing vascular skin lesions such as extensive hemangiomas, and may find a definite use for local destruction of certain types of cancers.

HYDROTHERAPY

The employment of water as a physical therapeutic agent depends principally on the effect of certain physical forces. Water has a great

for applying heat. Its utility as a thermal agent is further enhanced by the ease with which its temperature can be measured, regulated, and controlled.

Water can readily be applied with varying and regulated pressure. A body immersed in water is buoyed up by a force equal to the weight of the water it displaces. Extended use has been made of this principle in recent years to exercise under water muscles too weak to work against gravity out-

side of the water and to exercise joints stiff because of injuries or arthritis. Modern methods of water treatment can be classified under the headings of *hydrothermal* and *hydrokinetic* measures. In addition there are also hydrochemical effects obtained with some special forms of baths.

Hydrothermal Measures. The normal or average temperature of the skin surface is about 92° F. If water at a temperature different from that of the skin is applied to the body it will either conduct heat to the body or absorb heat from it. The differences in temperature act as a "stimulus" or irritation of the nerve endings of the skin.

TEMPERATURES OF WATER APPLICATIONS

Type of Application	Degrees Fahrenheit	Degrees Centigrade
Cold bath	40 to 65	4.4 to 18.3
Cool bath	65 to 75	18.3 to 23.8
Tepid bath	85 to 95	29.4 to 35.0
Warm bath	95 to 100	35.0 to 37.7
Hot bath	100 to 110	37.7 to 43.3

The greater the difference from the normal skin temperature, the more marked are the physiologic effects of hot and cold water applications. Short applications of intense heat or cold are equally stimulating.

of low vitality cold applications do not bring about such a reaction and are, therefore, to be avoided. It is evident that aged persons can be submitted to such a powerful general stimulus only when they have been accustomed to it, and in most cases the local application of less intense stimuli, such as compresses and local baths, is to be preferred.

Compresses may be applied at a constant temperature, hot or cold, or at an initially low temperature which is gradually raised. This latter form is known as a stimulating or Priessnitz compress. A *cold compress* causes contraction of the peripheral vessels and is for immediate relief in recent injuries, as well as in early stages of inflammation, to combat swelling, redness, and pain. Cold compresses to the forehead are useful in cerebral congestions of various origin. A *stimulating compress* (Priessnitz) is a cold compress covered with several layers of dry flannel so that they completely overlap the wet linen underneath. The mild vascular skin reaction of the stimulating compress is a convenient treatment measure in many acute congestive conditions. A *throat compress* is useful in sore throat, laryngitis, and tonsillitis. A *chest compress* serves in affections of the respiratory system such as bronchitis and pneumonia. A *hot compress*, kept on for one to two hours, will bring on an intense hyperemia and help to soften or disintegrate inflammatory congestion and also relieve pain and spasm.

Wet Packs. Cold wet packs are applied for sedation and antipyretic effect and hot wet packs for elimination. We distinguish between full and three-quarter packs, according to the extent and size of the pack. With the wet pack, the contact of the cold wet sheet with the skin causes reactive peripheral hyperemia. The heat released from the dilated blood vessels accumulates between the skin and the pack, forming a layer of warm air. A decidedly sedative and hypnotic effect is produced by the action of warm vapor on

sensory nerve endings, by depletion of cerebral vessels resulting from repletion of peripheral vessels, by the absolute immobility enforced by the snugly fitting pack and by the entire absence of mechanical stimulation. The pulse rate and respiration are slowed after primary stimulation. Repeated wet packs are antipyretic in effect.

Ablutions or sponge baths consist of pouring water over the entire body or part of it and serve as a mild form of thermal stimulation, especially suited for getting the aged patient accustomed to cold water and also for judging his vascular reaction.

Baths Baths may be classified according to their extent as full or partial baths and according to their temperature as cold, tepid and hot baths. A *full cold bath* or cold plunge elicits the strongest reactive response from the vigorous patient. It is generally contraindicated for the aged, as well as for those with high blood pressure, weak heart, or tendency to hemorrhage. A *continuous tepid bath*, administered at a temperature from 94° to 98° F, is one of the most effective sedative measures and is widely employed in mental hospitals to quiet disturbed patients. The physiologic effect of the continuous bath is a filling of the peripheral vessels with a corresponding depletion in the deeper parts, notably in the brain. Due to the bettered nutrition of the skin, indolent ulcers, suppurating wounds, extensive burns, and extensive skin lesions are influenced favorably.

A full tub bath at a temperature of 100° to 108° F is known as a *hot bath*. It brings about an increase of metabolism and elimination. In chronic arthritis and rheumatoid conditions hot baths at a temperature from 96° to 102° F, employed from five minutes to one half hour two or three times a week, are a most useful measure for home treatment.

Contrast baths consist of alternate immersion of a part for three minutes in hot water (105° to 110° F) and for one minute in cold water (60° to 70° F), repeating this several times. This performance causes an intense vascular reaction and may produce quite a strain in peripheral vascular disease because of the extreme vasoconstriction. Hence, contrast baths should not be used on patients with advanced circulatory disturbances, but they may be usefully applied on those with sluggish circulation, following wounds and scars, for the toughening of stumps and in their preparation for massage. They are useful also in the treatment of chilblains.

Special Forms of Baths Among special forms of baths are carbon dioxide baths, galvanic baths, and paraffin baths.

The carbon dioxide bath affects the blood vessels and the heart action, causing a dilatation of the peripheral vessels and an increase in pulse pressure, cardiac output, and blood pressure. At 92° F it facilitates the work of the heart, while at lower temperatures (never below 86° F) it increases its work. By regulating the temperature, duration, and frequency of the bath, as well as the saturation with carbon dioxide and salt, the effects on the individual can be varied judiciously. In various kinds of heart disease, both valvular and myocardial, favorable results can be obtained by a course of baths. *Medicated baths* containing aromatic, resinous, and other substances have received attention from time to time, but offer only a very limited scope of usefulness. Extravagant claims made as to supposed special benefits in rheumatic conditions have not been corroborated by any large scale clinical experience.

The paraffin bath consists of immersion of the extremities in melted

paraffin or the application of this paraffin with a paint brush to the surface of the body (Fig 38) The paraffin bath is useful in the after treatment of traumatic conditions involving the extremities with much swelling and stiffness and is also quite effective in chronic osteoarthritis of the hands On areas with disturbed skin sensation following nerve injuries and on recent thin scars the bath must be applied with great care It should not be used at all in cases of skin infections or open wounds

Hydrokinetic Measures In the *whirlpool bath*, water at a temperature between 105° and 110° F is kept in constant agitation in a vessel which holds

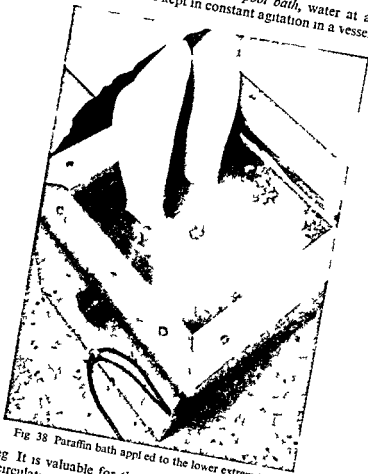


Fig 38 Paraffin bath applied to the lower extremities

the arm or leg It is valuable for the early treatment of stiffness pain and sluggish skin circulation following fractures In painful scars adhesions joint stiffness indolent wounds and mild forms of peripheral vascular disease it is also employed with benefit Care must be taken to avoid overheating and near collapse in older people by the prolonged use of hot whirlpool baths to the lower extremities it is best to apply them for only fifteen to twenty minutes instead of the customary half hour

Therapeutic pool treatment or *underwater exercise* takes advantage of the fact that the buoyancy of the water reduces the weight of the body enabling weak muscles to be exercised actively the heat of the water also relaxes

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sunburn, and such symptoms as headache, lassitude, undue fatigue, irritability, or a gastrointestinal upset

Solaria with glass transmitting ultraviolet rays are useful in hospitals and sanatoria where patients can be exposed, just as for outdoor sun treatment, but with protection against drafts and chilling. However, increased penetration for heat and light rays and lack of sufficient ventilation often make the atmosphere of these rooms as oppressive as that of a hothouse. It is also evident that when there is no appreciable ultraviolet present in the sunlight, as in the winter months, these windows are of no value.

In addition to the conditions amenable to heliotherapy, *artificial light therapy* is a valuable adjunct to general medical treatment in selected forms of general debility, in secondary anemia, in convalescence after operations and infectious diseases, in chronic bronchitis and sensitiveness to acute respiratory disorders.

Local ultraviolet irradiation is employed for its bactericidal effects in superficial infections, infected wounds, and in dermatophytosis, as well as for mild stimulation of sluggish ulcers (selected forms of varicose ulcers) and burns of various degrees. The admixture of thermal and visible radiation in these cases is helpful and the heat evidently plays a role in inducing the desired effects.

Contraindications to ultraviolet irradiation are advanced cachexia or inanition from any cause, advanced heart disease, with failure of compensation, advanced arteriosclerosis, and gross renal or hepatic insufficiency. In hyperthyroid subjects and patients with diabetes, severe itching and annoying general symptoms may occur after irradiation. Highly nervous people are often made worse and at times develop marked pruritus. All forms of generalized dermatitis, as a rule, serve as a contraindication to ultraviolet irradiation.

Choice of Ultraviolet Lamps. Ultraviolet generators can be classified in three groups: (1) lamps emitting infra red, luminous, and ultraviolet rays, such as the carbon arcs and the mazda sunlight lamps, (2) lamps emitting luminous rays and both long and short ultraviolet, such as the hot quartz lamps, and (3) lamps emitting chiefly short ultraviolet rays, such as the cold quartz lamps.

In making a selection of ultraviolet generators, the principal question is whether additional heat radiation is desirable. Heat in itself is beneficial in rheumatoid conditions and in joint and glandular tuberculosis. Therefore in these conditions carbon-arc irradiation may be preferable, or an infra red generator may be employed in addition to the "cold" ultraviolet source. For general tonic treatment in the aged, additional heat radiation is generally preferable. On the other hand, an excess of heat radiation is often undesirable in pulmonary or intestinal tuberculosis, hence the preference of a radiation restricted to ultraviolet in these conditions.

ELECTROTHERAPY

A comparison with a water system is the best introduction to an understanding of the physical characteristics of the principal electromedical currents.

COMPARISON OF WATER SYSTEM AND THERAPEUTIC CURRENTS

Steady flow, at low pressure

Squirts or waves, at low pressure

Heavy flow of extremely rapid waves at high pressure

Galvanic current

Low frequency currents

High frequency currents

The effect of an electric current depends on its kind and the mode of application. The effect of an electric current depends on its kind and the mode of application.

of electricity to a varying extent. Two principal biophysical phenomena may take place upon the passing of an electrical current: (1) movement of ions resulting in changes of ionic concentration; (2) increased vibration of molecules resulting in increase of temperature.

The *galvanic current* consists of an uninterrupted flow of electrons in one direction at low voltage. Through electrochemical changes it exerts a mild stimulative effect: a prolonged increase in blood supply and thus increased nutrition of the region affected. Galvanic baths also influence the general circulation and metabolism. Medical galvanism has proven clinically useful in a number of acute and chronic inflammatory conditions such as: (1) selected cases of traumatism, contusions, sprains, myositis; (2) selected cases of arthritis and rheumatic conditions, neuritis and neuralgia, mostly in the chronic stage. It should be remembered as a useful alternate of diathermy treatments, especially in heat sensitive patients. Medical galvanism often serves as a useful adjunct in the management of some circulatory disturbances of the brain such as in the after treatment of selected cases of cerebral hemorrhage. It can be employed with benefit in patients in the subacute stage with headache and dizziness, if not due to persistence of high blood pressure.

Ion transfer or iontophoresis consists of the introduction of medicinal ions or electrically dissociated particles of drugs into the skin or mucous membrane. It is brought about by the polarity effect of the galvanic current: ions with a positive charge are introduced from the positive pole; those with a negative charge from the negative pole (like electric charges repel each other). Ion transfer of *heavy metals*: solution of copper or zinc exerts a mild caustic effect and is useful in treatment of sluggish wounds and ulcers and in chronic infections of sinuses and cavities. Ion transfer of *vasodilating drugs*: histamine and mecholyl serve to improve the peripheral circulation in rheumatic and other conditions; the vasomotor action by the galvanic current enhances the effect. Histamine ion transfer acts favorably in traumatic and rheumatic affections of soft tissues, e.g., fibrositis, neuritis, traumatic arthritis. Mecholyl ion transfer may be useful in chronic rheumatoid arthritis, in peripheral vascular disease where spasm is a major factor, in Raynaud's disease, in thrombophlebitis and in varicose ulcers. Absorption of the drugs into the general circulation by overdosing or in case of special sensitivity is a definite possibility and calls for cautious administration.

Low Frequency Currents: Sudden make and break or gradual rise and fall

with an unimpaired nerve supply. Other low frequency currents are the surging faradic, the slow sinusoidal and the modulated alternating current. The galvanic and faradic current is employed in electrodiagnosis to furnish a picture of the gross anatomic condition of the weak or paralyzed muscles and to assist in determining the prognosis.

Electric muscle stimulation employs the form of low frequency current to which weak or paralyzed muscles respond best (Fig. 39). When considering

the treatment of such muscles there can be no argument on the point that voluntary exercise is the most desirable form of activity. When muscles are atonic and wasted from any cause, and voluntary exercise is not feasible, then, so long as the nerve path is intact, the production of painless graduated muscular exercise by electric stimulation reproduces the physical and chemical phenomena connected with normal muscular work. For muscles in flaccid paralysis, due to nerve injury, electrical stimulation helps to preserve at least part of the functional properties of the muscles until normal nerve impulses return. In spastic paralysis, following hemiplegia, electric stimulation may be useful as an adjunct to muscle reeducation and training. It is also an important aid in maintaining the patient's morale through the trying period of readjustment. It will, however, not restore voluntary muscle control.

Electric shock treatment by a low frequency current to the brain has proven increasingly useful in melancholias of aging patients, particularly following climacterium, as well as in other depressive psychoses.

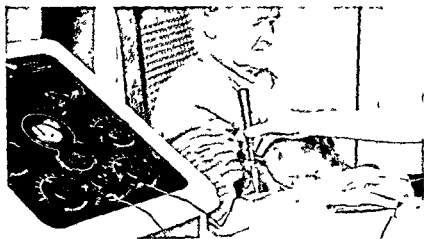


Fig. 39 Electric muscle stimulation of forearm with low frequency apparatus (International Clinics, 4 J. B. Lippincott Co.)

High frequency currents, in the form of long wave and short wave diathermy, do not cause any appreciable movement of ions because of their extremely rapid alterations. Their oscillating energy serves to increase the internal vibration of molecules in the path of the current, resulting in an increase of local and, under suitable conditions, general body temperature. Short wave diathermy requires a less exacting technique than the use of either air spaced electrodes or the inductance coil. Hence its widespread adoption in recent years. The most recent form of high frequency heating consists of still shorter waves of the radar type (microwaves) which do not require the application of electrodes or pads near the skin and penetrate effectively to a depth of several inches.

The essential clinical effects of all forms of diathermy are deep hyperemia, promotion of resorption of inflammatory exudates and adhesions, and relief of pain. This often results in the decrease of swelling and restoration

of function in the treatment of subacute and chronic inflammations of joints and bursae. It may also be effective in congestive lung conditions and in chronic inflammations of abdominal and pelvic organs, such as the gall bladder and the prostate. The symptoms arising from peritoneal adhesions and spastic conditions of the abdominal organs (stomach, gallbladder, intestines, and pelvis of the kidney) are often alleviated by diathermy.

The contraindications and dangers of diathermy in the aged must be considered carefully, especially in those with impaired sensation of the skin, occlusions of blood vessels, and other conditions. In such cases, safer methods of superficial heating are satisfactory. Ordinary contusions, abrasions, and other skin lesions should be avoided.

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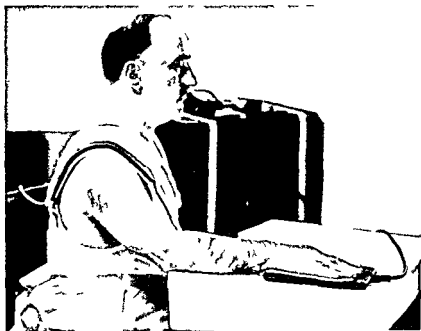


Fig. 40 Short wave diathermy along upper extremity (Kovács: *Electrotherapy and Light Therapy*, Lea & Febiger.)

myositis, many rheumatic conditions readily respond to radiant heating. In acute inflammatory conditions and in hemorrhagic tendencies, diathermy is absolutely contraindicated. Diathermy must be used cautiously at all times and in small doses, because of danger of overheating tissues. A useful substitute in many instances is the galvanic current.

Electrodesiccation or the destruction of diseased tissue or accessible small neoplasms by the drying effect of the monoterminial high frequency current is a convenient and simple method in many annoying conditions of the skin and mucous membranes. Warts, moles, papillomas, and other benign growths of mouth, the nose, and vagina can be easily removed with no hemorrhage and with good cosmetic results. Care must be exercised in precancerous lesions that destruction be thorough.

MECHANOTHERAPY

Mechanotherapy comprises massage and therapeutic exercise

Massage. Massage is one of the simplest and most useful forms of physical treatment and when correctly applied will give many aged people great comfort. The physical elements constituting the different movements of massage are (1) contact between the skin of the operator and the skin of the patient, and (2) pressure, the application of a varying amount of mechanical energy to the touching surfaces. The effectiveness of the treatment depends by no means on the amount of physical effort expended. In applying massage, three main varieties of movements are employed singly or in combination: stroking, compression, and percussion.

Stroking, or *effleurage*, is the fundamental form of massage, which is almost instinctive. The mother gently strokes a child's bruised forehead with the palm of her hand in order to relieve pain and to diminish swelling. Patients stroke aching parts in seeking to ease their painful sensations. Every massage treatment starts with stroking. This is the form of general massage which is most beneficial, because of its sedative effect, in insomnias, indefinite aches and pains of aged people, and for its gentle stimulating effect on circulation. Many persons go to sleep more easily after a general body massage.

The local use of the more stimulating forms of massage is indicated in the after treatment of traumatic conditions, in chronic osteoarthritis and fibrositis. In spite of its popularity in "beauty culture," massage does not remove the wrinkles of old age, neither can it disperse nor squeeze out lumps of fat under the skin.

Massage is often beneficial to the aged when they are not able to exercise daily. In conditions of muscular weakness which are due to disuse, general systemic inactivity, or exhausting disease, massage will help to keep up muscular tone and prevent further atrophy of the muscles. Edema of the skin and subcutaneous tissue, due to circulatory changes, can be locally dispersed and the work of the heart relieved by combined local and general massage. The flow of blood becomes accelerated without straining the heart, edema, cyanosis, and shortness of breath are relieved. In chronic circulatory diseases due to a weak heart muscle, massage is a most beneficial measure at the bedside. Atonic conditions of the gastrointestinal tract, especially chronic constipation, can be considerably benefited by appropriate massage. Massage, properly applied, may be considered as vicarious exercise without fatigue or strain.

Contraindications and Dangers. Wounds, skin eruptions, and inflammatory processes in or underneath the skin preclude the use of massage on the affected parts. Acute inflammatory conditions anywhere in the body, but especially in the abdomen, are also contraindications, likewise tumors and tuberculous joint affections. In the acute stage of local inflammatory and irritative conditions of the peripheral nerves, neuritis and neuralgia, massage is as a rule harmful, for even mild stroking may bring on a violent increase of pain. Arthritic joints being already a seat of an inflammatory process, it is a cardinal rule that massage in arthritis must be gentle and must be carried out in the neighborhood but not immediately over the affected joints. Massage should not be employed on the lower extremities of patients with arteriosclerotic impairment of the peripheral circulation (see p. 450). A reduction

in the amount of massage should be made in all cases of senescence. Proper rest after all forms of general massage is essential.

Therapeutic Exercise. Dysfunction of many organs is often due to muscular inactivity and habitually poor posture. Therapeutic exercises may be either *general*, in which the organism as a whole is affected, thus acting upon any special organ indirectly, or *functional*, to repair dysfunction of an organ. For curative purposes exercises are also subdivided into passive and active. *Passive exercises* consist of massage movements and passive movements at the joints. They are carried out by a technician or by the weight of the patient's body. While of service in certain conditions, such as stiff joints

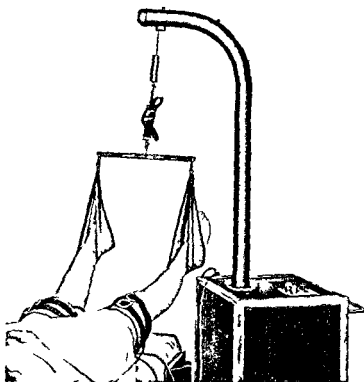


Fig. 41. Combination pressure and exercising device for peripheral vascular disease (Plethorator Co.)

caused by contracted ligaments and muscles, they lack the stimulating effect of active exercises. *Active exercises* may be single or duplicated and these latter may be *assistive* or *resistive*. The physiologic effects of these movements differ considerably. Hence it is necessary to know the pathologic changes in a given case in order to prescribe exercise intelligently and to insure that the prescription is properly carried out.

In most people not engaged in active outdoor life the daily use of some simple form of setting up exercises is desirable in order to keep fit and to be able to take up outdoor games when opportunity permits. Depending upon the strength and condition of the individual, these may be varied from simple exercises, taken lying down, to gradually increasing activity while sitting and

then standing and running in place. Medical gymnastics are best taken in conjunction with heat, massage, hydrotherapy, etc. Heat, either wet or dry, is the first procedure. Massage may follow the heat or may end the exercise seance. *General exercises* often may be usefully employed in derangements of the circulation, respiration, and digestion. They are especially useful in the following conditions: constipation, enteroptosis, gastric or intestinal neurosis, chronic passive congestion, compensated cardiac valvular disease, compensated myocarditis, arterial hypertension, arterial hypotension, atelectasis, postoperative empyemas, and chronic pleuritic adhesions. *Functional exercises* will be particularly concerned with traumatic lesions such as fractures and dislocations and their sequelae, deformities of joints and of the spine, nerve disturbances such as paralyses, the arthritides, and in peripheral vascular disease.

Contraindications to exercise include, in addition to inflammations or active infections, any depleting disease or new growth. *The condition of an aged patient's heart must determine the allowable amount of general exercise.* Both dizziness and fatigue after exercise indicate that the cardiac reserve is lessened. All sudden strains on the heart in old persons must be avoided.

If old people insist on exercising as freely as they did in their younger years, this may be condoned if they are used to that form of exercise, and it causes no symptom of strain. As a general principle, however, exercise in the aged is best taken daily, in small amounts. (See Chapters 6 and 7.)

Occupational Therapy. Occupation as part of a physical therapeutic regimen has received widespread use in recent years. Its object is to restore function and the work habit; its two main forms are diversional or recreational and functional. In patients with senile psychosis, for instance, some particular activity or work under skilled direction and within the mental capacity of the patient will counteract deterioration; in the after treatment of traumatic conditions occupational therapy will greatly facilitate return to normal activity and, together with early corrective exercises, will prevent muscle atrophy and loss of muscle power in the good muscles and deterioration in the body as a whole. Modern occupational therapy has become real exercise therapy in many respects and should receive careful consideration.

INDICATIONS FOR PHYSICAL TREATMENT

Among conditions in the aged in which physical therapy is most frequently indicated and promises benefit are: (1) rheumatic conditions—osteoarthritis, gout, fibrositis, myositis, and neuritis, (2) cardiovascular ailments—arteriosclerosis, hypertension, and resulting myocardial weakness, also peripheral vascular disease, (3) nervous and mental ailments—hemiplegia, senile psychoses, and other degenerative conditions, (4) genitourinary conditions, prostatic and pelvic involvements, (5) the large group of traumatic conditions—in these as well as some of the foregoing, physical medicine may serve for local treatment as well as for general body conditioning.

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CHAPTER 11

MEDICOLEGAL ASPECTS OF SENILITY

A WARREN STEARNS

THE course of life is sometimes spoken of as its trajectory. The individual starts at birth with certain organic drives which motivate him, with certain capacities for growth, and little else. Gradually the force of the initial impetus given him by his basic drives is directed along certain lines by the parents. This is called training. Later the child is taught certain conventional things about life, human experience, and accumulated knowledge. This is called formal education. Then come the adult problems of "getting" and "begetting," that is, the individual must earn a living and after mating, build a home and rear a family. Following this come the rich years of maturity with their accomplishments and satisfactions. Finally, evening comes with its declining powers and abilities. Life ends as it began in utter helplessness.

Now man does not live in a vacuum but in a medium peopled with other lives with which he has mutual responsibilities, that is, society expects the trajectory of life to be somewhat conventional and holds the individual by its mores to patterns of behavior which are socially acceptable. The law takes cognizance of the behavior of individuals and is that department of society which attempts to regulate the grosser vagaries of conduct and help maintain a degree of conformity consistent with the commonweal.

LEGAL INCOMPETENCE

In Childhood. Since the dawn of history the incompetence of children has been recognized. Until a child has reached an appropriate age, he or she cannot be held for crime, cannot marry, cannot hold title to real estate, and is automatically disenfranchised. However, when he has reached the age of twenty-one, he is considered to be fully mature and is said to be "competent." The burden of proof is on anyone who questions his competency. This full legal responsibility lasts until the end of life unless it is challenged and incapacity demonstrated by positive proof.

In Old Age. Yet the relative helplessness of older people has long been recognized by the law. Until recently children have been held responsible for the support of aged parents. Old age assistance, although the emphasis has been laid on physical disability, has perhaps been more often needed because of mental disabilities. Every one having to do with aged persons recognizes their limitation of responsibility, although the law holds them completely responsible. Their children or caretakers gradually return them to the discipline of childhood by pampering in some cases, by harshness in others. The corner is turned several times in life. *Prior to adolescence the child does not question the authority of the parents. Then there is a struggle for supremacy, but as the parents get older, the children gradually take over the rule and at last their relative status is absolutely reversed.*

If an old person's mind is affected to an extent that he may be said to be insane, this fact can be established and he is handled like any other person having a similar handicap. For this reason I shall not go into the handicaps of the insanity of old age but shall try to cover a few less definite and well defined disabilities (See Chapter 17)

A recent survey of a large number of elderly people has impressed the writer with the fact that very frequently the vicissitudes of later life are merely the culmination of the indiscretions of earlier years

DETERMINATION OF LEGAL RESPONSIBILITY

The question of criminal responsibility is a difficult one at best. The law must settle questions beyond the border of exact science. A person old or young is either one hundred per cent responsible or one hundred per cent irresponsible. Unless an aged person can be said to be insane in the ordinary sense, that is, committable, he is ordinarily not excused for crime, but is held to be fully responsible. When convicted, his infirmity is sometimes recognized in the disposition of the case.

Role of Family. When an old person's helplessness is recognized by his relatives there is legal provision for his protection through a guardian or conservator. The latter merely has charge of the property of the person while the former has charge of the person and the property. Often the barn is locked after the horse has been stolen. Relatives are loath to apply for guardianship for fear of offending their loved one. Old people resent supervision or direction by their children or relatives and tend to turn against anybody who is forced by necessity to take charge of them. As a corollary to this they turn toward those who agree with and flatter them. Aged persons tend to be penurious in the ordinary affairs of life, but give freely to courtiers, sycophants, and self seekers. Many large estates have been entirely dissipated, and the cupboard is bare when in desperation the children have at last applied for guardianship. Neighbors and casual friends see only the superficial alertness of the aged person and stoutly maintain that he is all right. It is often difficult for honest relatives to establish incompetency.

Role of Physician. The medical, as well as the legal profession has a great responsibility in these matters. Often the doctor forms a casual judgment based upon purely sentimental grounds. He takes sides either with the children or with the old people and rarely goes to any length to determine whether or not there are mental changes indicating the need of a guardian or conservator. Many old persons die in the poorhouse or on charity because a mistaken kindness on the part of their children has postponed legal action until it is too late. An impaired memory, a vacillating will and an unstable emotional life readily point the way to those minor changes in mind which so often result in gross changes in conduct.

Disposition of Property. One of the unfortunate facts of life is that judgments of the greatest importance often have to be exercised at a time when competency is questionable. I refer to the disposition of property by will. The lists of contested wills before our courts are tremendous and, although they often represent fighting out old feuds begun before the death of the testator, such cases are frequently the beginning of a feud which is to last for generations to come. Where there is perfect harmony among near relatives the task is often done with perfect understanding between all in spite of great

incapacity on the part of the testator. Where there is distrust and suspicion among relatives, where the person involved is alone and not too closely acquainted with legal heirs, the seeds of discord are already sown in the greed inherent in human nature. The law recognizes that a person has a right to say who shall inherit his property, provided he has testamentary capacity. In a rough way this consists in knowing the nature and extent of his property, in having a knowledge of those who have a claim to his bounty, and sufficient mental power to act upon such knowledge. Obviously, there is no test of capacity and although the courts claim to be trying the question of the man or woman's mentality, they are often trying the will itself and serve as referees in a quarrel between warring factions.

It sometimes seems that it might be wise to set an age beyond which inheritance should take place by statute, just as the legal responsibilities of children are not fully recognized until the age of twenty one. Old people, although not insane, are susceptible to flattery. Woe to the heirs of the silly, old man who has an artful and beautiful young friend. With the advent of public care of aged persons the children have been released from their responsibilities. Likewise, aged people have been liberated from any degree of control by their children. This among other causes has led to the growing up of innumerable rest and convalescent homes of varying degrees of altruism and wickedness. Often the aged person with a little property, helpless and living in such a place, is sentimentally moved toward the hand that feeds and caresses. The rights of the legal heirs present a more or less academic question, but the emotional response arising between the aged person and the one who cares for him by day and night is a concrete experience. Slyly a lawyer is brought in a perfect stranger, perhaps, a doctor is standing by who naively says that the old man seems all right to him, a will is made, sweetened perhaps with a little gift to the church. Then the old person dies and the children hurry to the court to present their will. Alas, it is too late as a later will has been presented. The children are often left to pay the bills, so cleanly has the old person been stripped of his fortune. There is no cure for this except to establish universal honesty on the face of the earth. Wherever there is human weakness, there will be human artfulness to take advantage of it. Needless to say, old people with failing powers should be carefully nursed by honorable persons. Their relatives should see to it that no one has cause to come between members of the family. Lastly, if a doctor or lawyer is asked to participate in making a will, he should assume a judicial attitude and not accept partisan, malicious statements against heirs or others with legitimate interests. *More important still, there should be some survey or scrutiny of the mental powers of the individual before a will is executed.*

In order to make a legal contract the parties must be of sound mind. As noted above, old age needs protection. Distant pastures look green to all of us and more particularly to old persons. Many times old persons are induced by predatory persons to part with their property by an agreement to take care of them during the rest of their life. The property is soon squandered and the old person's feebleness requires a lot of care. The property well managed would have furnished this care but, having been squandered by these false friends, the person now has no recourse. Old persons, because of their incapacity, are unable to care for their affairs and so are often led to make contracts which ultimately prove very disadvantageous to them.

Marriage of Aged Marriage itself is a contract and a whole chapter might be written on the vagaries of old persons at the altar. Old men often marry young women. Less frequently, young men marry old women. The law tends to be liberal in such matters and in my experience it is very difficult to get a marriage annulled, once it has been consummated. When an old man marries a young girl it does not represent the type of affection which is usually designated as married love. The old man wishes either a toy or a pet, or, perhaps, a housekeeper. What does the young woman want? Perhaps a father or an older brother, but more often a bank account. This leads to all sorts of unhappiness. It is astonishing how easy it is for utterly incompetent people to get married. It is likewise astonishing how difficult it is to get them separated by law. I am frequently consulted by the children of aged parents after such a marriage has taken place. Alas, the time to have done this was when the marriage was imminent. However, children often do not know that the marriage is contemplated or that it has been consummated. The marriage of an aged person to a young person always raises the question of mental incompetence or bad faith. How much mental power does one need to make a marriage contract? In my experience very little is needed. On the other hand, if pretended love affairs involving aged persons are seen by their relatives, the mental state of the old person should be carefully scrutinized and a guardianship seriously considered. With the money protected, the aged prey seems much less attractive.

Suicide grows progressively more frequent as age advances. While of itself it does not betoken insanity, in a large percentage of cases it does indicate mental disease and may have very important medicolegal complications. Suicide by an aged person may bring a previous will into question. It may likewise bring a previous marriage or other contract into question. It frequently involves the extent of life insurance as well as any other act the legality of which it is possible to question. Most of the senile suicides have melancholia, though a large number have either arteriosclerotic or senile dementia. In a few it appears to be a more or less rational act in the face of a somewhat severe threat, such as cancer. Generally speaking, where an aged person commits suicide there should be a serious investigation as to his previous mental competency.

OLD AGE AND CIVIL PROCEEDINGS

Accidents By far the most frequent proceedings in court are actions of tort. These represent claims for personal injury, and here again, old age is a common battleground. In the first place, old persons, because of their helplessness, are injured more frequently and it is often a fine question as to how much contributory negligence there is in the case, thereby decreasing the liability of the defendant. Aged persons often wander out into the streets on ice and snow and become involved in accidents which do not in any way represent carelessness on the part of the plaintiff. For this reason, it is very important to learn in great detail the mental status of aged persons and their habits.

Perhaps the accident has not been serious in itself but serves as the precipitating cause of senile disability. The relation between a shock or

cerebral hemorrhage and an accident is a knotty problem with much difference of opinion among equally competent and honest doctors. Although a person has had a high blood pressure, if he is involved in a serious accident, with or without a head injury, and almost immediately has a cerebral hemorrhage or thrombosis, it seems to me fair to believe that the accident bears a causal relation. If, on the other hand, a person has had a previous cerebral accident, receives a minor injury, and then weeks or months later has another cerebral accident, it is folly to relate the two, although in the minds of the people themselves there is a close connection. It is one of the common traits of human nature to associate any untoward event with any catastrophic experience which has taken place at any other time in our life. Needless to say, attempts to get aged persons to sign releases for damage in accidents for trivial amounts are vicious and should be thoroughly scrutinized by responsible relatives. When mental impairment is found, the presence of arteriosclerosis is often an adequate explanation. The presence of arteriosclerosis in an old person is no indication whatever of impaired mentality.

CRIMINALITY IN OLD AGE

Premonitory Signs of Senile Dementia. Oftentimes the breaking through of inhibition and reserve on the part of an aged person is a very early evidence of senile dementia, although a little time is needed before the well recognized memory defect makes dementia obvious. Furthermore, unskilled persons are often deceived by superficial alertness. If, in the beginning of senile mental changes, there is confusion or dulness, it is readily recognized. While there may be a marked impairment of recent memory, and while there may be moral blunting of extreme proportions, if there is a superficial alertness relatives and friends usually interpret this as a well preserved mind. The hilarity and jocularity of the aged is evidence of decay, although such decay does not amount to senile dementia. A well preserved personality in old age maintains the same standard of aesthetic sensibility, moral perception, emotional control, judgment, and so on, which he has had throughout life. It is not reasonable to have two standards of competency, one for young persons, another for old persons.

Minor Transgressions. There is an irascibility and irritability which frequently comes during advanced age, especially with ill health. The pugnacity arising from these often results in legal manifestations. Assaults and even murders sometimes ensue. Again, where there is a depression due to advancing senility with its enfeeblement or, perhaps, deafness and/or blindness with economic stress it is often very difficult to know whether one is dealing with a reaction to pressure or a definite psychosis.

Violation of sanitary codes indicating aesthetic blunting is not of infrequent occurrence. Boards of health have a great deal of trouble with old people whose untidiness and filth in caring for garbage, cesspools, and animals is proverbial. Also, the morbid attachment to animals and pets is well known. The police courts frequently have to deal with old ladies so impoverished they can hardly feed themselves, who have innumerable cats and dogs, and create a nuisance with their excessive devotion to pets.

Old persons frequently show a decreased tolerance to alcohol and this results in irregularities of conduct which bring them before the criminal courts.

Impairment of integrity and honesty frequently leads to criminal situations. One often sees a trustee whose life has been a model of integrity, yet who long before senile dementia could be diagnosed commences to pilfer from his clients. He oftentimes loads himself up with gold mine stocks, horse-racing bets and similar get-rich-quick devices. As his business sagacity and capacity commence to decline, he is more apt to seek excitement and success in questionable enterprises. Presenting an external appearance of benignity and wisdom, such persons have often involved their wards in hopeless financial difficulties. Sometimes petty thieving appears as an early symptom of senile changes in persons hitherto scrupulously honest.

Sexual Transgressions. Nothing is more pathetic than to see an aged person brought before the bar of justice accused of crime. Frequently one sees aged persons involved in criminal proceedings where there is no serious question of insanity, yet where it appears obvious that their delinquencies are associated with the deterioration of later life. In the sexual field, as physical vigor is lessened there is a greater interest on the part of older people in unusual sexual experiences and especially in young persons. Carnal abuse of a female child often represents an attempt on the part of an older person to stimulate some fleeting remnant of sexual desire. Furthermore, when a person is relatively incompetent sexually, there is a certain embarrassment about engaging in sexual affairs with mature persons who know what it is all about, although experiences can be had with children whose innocence saves the face of the older person.

The handling of young girls by older men and the maudlin attitude of such persons toward them, often condoned by their parents as a fatherly or grandfatherly attitude, needs but one step to constitute a criminal interest, and is, in fact, a precursor to a criminal interest. The families of the aged often deny any ulterior interest and defend the action of their aged person by saying he has been impotent for many years. Many vulgarisms currently afloat show the extent to which the dangerousness of the impotent person has been traditionally recognized. There may be considerable sex interest and activity despite complete impotence. When older men fondle young children lasciviously, whether male or female, it is evidence of a beginning deterioration, as well as the release of a lascivious interest. The affairs between old ladies and so-called gigolos are in the same field, although they are often less obvious.

CONCLUSION

Finally, although the law provides protection and a remedy for some of the vicissitudes of old age, it still remains an obvious truth that in the long run the aged are quite dependent upon the integrity, good faith, and honorable dealings of those who have them in their care. Since government has now assumed so large an interest in the problems of the aged, it is obviously necessary that thorough case work be carried out with such beneficiaries in order that they may get the fullest advantage of aid and be protected from exploitation by relatives or other persons.

SECTION II

DISORDERS OF METABOLISM

CHAPTER 12

MALNUTRITION

WILLIAM H. SEBRELL

INTRODUCTION

NUTRITIONAL problems are usually more difficult to handle in the aged than in other age groups. The eating habits of a lifetime are not changed easily and patients may rebel against too much restriction or too wide a change from accustomed foods, or new methods of food preparation which may be desirable if health and efficiency are to be maintained. The nutritional guidance of the aged involves much more than merely devising adequate diets.

Proper nutrition is one of the most important problems confronting the physician who is treating the aged. The constant or repeated insults to the organism of inadequate or improper nutrition have cumulative effects through the years and at least part of the damage from such nutritional defects may be irreparable. The prevention of such occult damage during senescence is even more important than the treatment of acute deficiency diseases. Prophylactic guidance in dietetics during maturity constitutes a valuable approach to preventive geriatrics (see p. 117). The influence of the nutritional practices of early life extends into old age and the measures to prevent degenerative diseases should be started in early life. Proper nutrition beginning early in life is an important factor in determining the number of useful years of life.

There are a number of changes in the aging individual which should be taken into consideration in dietary planning. The more important of these are the decreased metabolic rate and lessened activity which decrease the caloric requirement, the general decrease in tissue fluids, the changes in digestive secretions, and the decreased rate of absorption which may increase the requirements for certain other nutrients such as the vitamins. The increased susceptibility of the aging nervous system to adverse environmental changes is one of the factors which makes the maintenance of an optimal diet by the aging person a very important matter.

In considering nutritional defects we must take into account overnutrition as well as undernutrition.

OVERNUTRITION

It has been suggested frequently that many of the ills which beset the aging and the aged are the result of overnutrition and its consequent obesity.

Atherosclerosis, diabetes mellitus and senile cataract have been ascribed to habitual overfeeding of lipids and carbohydrates

Excessive Calories Experimental evidence indicates that feeding diets restricted in calories prolongs life and delays the onset of senescence. However, although no virtue has been assigned to the continuous feeding of diets of high caloric value the inclusion of a high proportion of foods of good vitamin, mineral, and protein content has been found beneficial, as has the feeding of high absolute amounts of certain individual vitamins. A diet excessive in calories is not necessarily adequate in vitamins or minerals. There can be no doubt that obesity is decidedly antagonistic to longevity.

Excessive Lipids Fatty livers and atherosclerosis have been ascribed by some clinicians and investigators to excessive amounts of neutral fats and cholesterol in the diet. Best and his associates¹ found that the production of fatty livers by high fat diets could be prevented by the inclusion of ample choline in the diet.

Cirrhosis of the Liver Work with experimental animals has shown that cirrhosis can be produced by feeding a diet low in choline. Patek and Post² report good results in the treatment of cirrhosis of the liver by the use of a diet containing about 140 gm of protein (including 50 gm of brewers' yeast) moderate fat, and totalling 3000-3500 calories. Their patients received injections of liver extract 5 cc twice weekly, and of thiamine chloride, 5 mg daily.

Other clinical studies have shown apparent benefit in cirrhosis by the use of choline with high protein low fat diets.³ Choline chloride has been given in oral doses of 1.5 to 6 gm daily. It should not be used parenterally. The most that can be expected appears to be some amelioration of symptoms and prolongation of life. The final prognosis is not altered.

It appears that liver function tends to become abnormal with increasing age. In a study by Rafsky and Newman⁴ 86 per cent of the subjects over sixty years of age showed at least one abnormal liver function test. These same observers have also found ether soluble red pigment in urine in a group of patients with liver disease.

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and wheat flour. It is notable that the high content of purines in the majority of these items makes their liberal administration inadvisable whenever the potentiality of gout exists. The association of gout with vascular sclerosis is well known (see p. 242).

Arteriosclerosis The relationship of diet to arteriosclerosis was discussed at considerable length by twenty-four pathologists under the editorship of Cowdry in 1933.⁵ Later work has been presented by Brody,⁶ Leary,⁷ and others. Although there has been considerable disagreement as to the part diet plays in the production of atherosclerosis and other syndromes, considerable evidence has been presented tending to show that, both under experimental conditions and under clinical conditions existing in modern times, foods rich in cholesterol and high fat diets have increased the incidence of arteriosclerosis, with or without diabetes, and of certain cardiovascular and renal injuries. Cholesterol rich foods are brain, eggs, butter, and milk.

Excessive Carbohydrates The influence of heredity in diabetes is important, but both clinical and experimental studies and statistical reports

support the view that one reaction of the organism to the overfeeding of carbohydrates, and possibly also excessive ingestion of fats is degeneration of the islands of Langerhans^{8 9} (see p 221) Mitchell and her associates¹⁰ have produced experimental cataracts by the feeding of excessive amounts of lactose or galactose. In the aged an excessive intake of both fats and carbohydrates should be avoided.

Excessive Protein. The dangers from the ingestion of excessive amounts of protein especially in patients with impaired renal function appear to have been overemphasized in the past. Although the experimental results are conflicting there is much evidence that a high meat diet does not cause kidney damage.¹¹ Under normal conditions the protein intake should approximate 1 gm per kilogram of body weight daily. If loss of albumin is occurring through albuminuria, sufficient protein should be given to make up for this loss unless there is excessive nitrogen retention (see p 592).

UNDERNUTRITION

Restricted Calories. McCay and his associates¹² have accumulated considerable experimental evidence that restriction in food intake increases the life span and delays the onset of senescence in rats. Saxton¹³ reports that the life span of rats can be increased by about 50 per cent through the regulation of nutrition. The method of regulation has been a high quality diet complete in all essentials, but restricted in calories.

In restricting the caloric intake of the aged to a level where a normal body weight will just be maintained special care must be taken to see that the vitamin and mineral intake remains adequate. The more the diet is restricted the more important it becomes to insure an adequate intake of these substances or deficiency symptoms may appear.

Protein Deficiency. This is likely to be overlooked as a contributing factor to ill health in the aged. Severe protein deficiency manifests itself by tissue wastage, anemia and edema. The diagnosis is easily made by the finding of a low plasma protein level. In increasing the protein intake a large proportion should be derived from animal sources such as liver, milk, meats, cheese, eggs, etc. Milder forms of protein deficiency must be suspected in mild anemias, habitual fatigue and apokamnosis. Detailed inquiry into dietary habits is very important with the elderly.

A negative nitrogen balance may be produced by serious burns, severe injury or extensive surgery. In these cases a large intake of protein is necessary to overcome the excessive loss. Protein hydrolysates may be used orally or parenterally unless there are contraindications.

Vitamin Deficiency Diseases. In considering the possibility of vitamin deficiency symptoms in the aged it is especially important to remember that deficiencies may occur in the presence of an adequate intake of vitamins by mouth if any condition is present which interferes with their proper assimilation. Diarrhea, vomiting, colitis, achlorhydria, alimentary malignancy and a host of other conditions may cause vitamin deficiency despite an apparently adequate oral intake. Under such circumstances an increased intake or even parenteral administration of vitamins may be necessary for correction of the deficiency. In the treatment of deficiency disease an adequate diet *must* be forced in all cases along with the administration of large doses of the necessary vitamins. This is very important, since such patients usually suffer from

multiple rather than single deficiency states and an adequate supply of a variety of foods is needed. Anorexia introduces a vicious circle into the problem unless the *necessity* of eating adequately is stressed.

The especial importance of vitamins to the aged is indicated by the work of Stephenson, Penton, and Korenchevsky,¹⁴ who noted a beneficial effect on some of the pathologic features of senility by the administration of a vitamin B complex preparation and vitamin C. They point out the importance of the prevention of partial or latent vitamin deficiency "in the approach to a less pathological senility."

Vitamin A Deficiency This may result in night blindness and yellowish, foamy thickened patches (Bitot's spots) on the bulbar conjunctiva. A dry folliculosis with hard keratotic plugs projecting from the hair follicles may also be found, especially on the extensor surfaces of the arms and thighs.

Sherman and his associates¹⁵ have found that adequate vitamin A deferred old age and increased the length of life of experimental rats. There is evidence that low grade deficiency reduces the body's resistance to infection.

TREATMENT 25,000 to 50,000 I U (International Units) of vitamin A daily. It may be necessary to continue treatment for several months.

Foods rich in vitamin A and carotene are the fish liver oils, liver, carrots, yellow squash, yellow sweet potatoes, spinach, kale, and the other yellow and green leafy vegetables.

Thiamine (Vitamin B₁) Deficiency This deficiency deserves special consideration in dealing with the aged patient because in advanced deficiency the enlarged heart, disturbed electrocardiogram, and edema of the extremities may create a misleading appearance of cardiac disease with decompensation. The loss of ankle jerks, painful calf muscles, and right-sided heart enlargement should aid in making the differential diagnosis. Milder degrees of deficiency are perhaps of very common occurrence and may manifest themselves by slight paresthesias and vague muscle pains. Especially common is tenderness of the calf muscles.

Recent studies also indicate that slight degrees of thiamine deficiency may produce symptoms similar to those of a neurosis in which the patient is depressed, loses interest in his surroundings, and becomes uncooperative (see p. 268). Frequently anorexia and decreased intestinal motility are added to the syndrome.

Rafsky and Newman¹⁶ have found that, apparently due to poor absorption and assimilation, relatively large amounts of thiamine (20 to 90 mg.) were necessary to saturate older people.

TREATMENT Thiamine, 30 to 50 mg. daily in 10 mg. doses, is given. Parenteral administration may be necessary until the acute symptoms subside. Then the patient should be carried on a maintenance dose of pure thiamine.

spongy, or bleeding gums unless the patient is edentulous. There also occurs increased capillary fragility and a tendency to hemorrhage into the skin following very slight injury. The finding of a low plasma level of ascorbic acid indicates a depleted ascorbic acid reserve. However, such diminution in the plasma ascorbic acid does not necessarily cause the symptoms of scurvy.

in every instance. With aging, the various organs contain decreasing amounts of vitamin C.

TREATMENT Ascorbic acid, 100 to 200 mg daily. Foods rich in ascorbic acid are oranges, grapefruit and other citrus fruits, tomatoes, and raw cabbage. Other fruits and vegetables contain ascorbic acid, but their contribution of this vitamin to the diet is normally negligible. Potatoes can be made an important source of ascorbic acid, if properly prepared.

Niacin Deficiency In addition to the usual picture of pellagra, there are two manifestations of niacin deficiency that are of special importance in the aged. One is a very mild condition, frequently overlooked, which is characterized by a smooth reddened tongue with or without digestive disturbances and a minimum of skin lesions. The other is a severe encephalopathic type of acute deficiency in which there are no skin lesions and the patient is stuporous or disoriented and confused and may have hallucinations, cog-wheel rigidities and sucking reflexes, thus presenting a picture similar to a toxic psychosis or exhaustion delirium. Such patients usually die unless energetic therapy with niacin is started promptly. Meyersburg¹⁷ has pointed out that senile psychosis may be simulated by pellagrous encephalopathy of the aged.

TREATMENT In the very mild cases niacin may be given in 50 mg doses twice to four times daily. A harmless reaction, characterized by flushing and a burning sensation on the skin, usually follows continued administration of niacin in small doses or after one large dose. The reaction usually disappears in less than an hour but the patient should be warned to expect it. Such reactions are rare if nicotinic acid amide, which is equally effective therapeutically, is used.

The encephalopathic type of deficiency may require a total daily dose of 1 gram or more of niacin. In severe cases it is advisable to give slowly 300 mg of sodium nicotinate intravenously.

Foods rich in niacin are lean meats, liver, salmon, and green leafy vegetables.

Riboflavin Deficiency Deficiency of this heat-stable factor of the vitamin B complex is characterized by fissures in the angles of the mouth, reddened lips, seborrheic accumulations on the folds of the body, and growth of capillaries in the cornea, which may be accompanied by corneal opacities. In the aged it is especially important not to confuse the fissures in the angles of the mouth with similar-appearing lesions seen in edentulous patients with ill fitting dentures or with buccal neoplasms (see p. 500).

TREATMENT Riboflavin, 10 to 15 mg daily by mouth, is used. Foods rich in riboflavin are milk, liver meats, eggs, and green leafy vegetables.

Folic Acid (Pteroylglutamic Acid, Vitamin B₁₂) This member of the vitamin B complex has been shown to be very effective as a therapeutic agent in sprue and in all forms of macrocytic anemia. It appears, however, that the spinal cord lesions in pernicious anemia may progress under folic acid therapy. For this reason, it is not to be regarded as a complete substitute for liver extract in therapy. A daily oral dose of 15 mg to 25 mg of folic acid should be adequate.

Vitamin B₁₂ The isolation of vitamin B₁₂ from liver furnishes a new substance for treatment of pernicious anemia, sprue and possibly other

macrocytic anemias. This substance may also improve the spinal cord lesions. The exact dosage has not yet been determined. A single intramuscular injection of 3 micrograms has produced a hematologic response in pernicious anemia.

Vitamin D Deficiency The requirements for vitamin D in relation to age are unknown. There is no experimental evidence on which even an approximation may be based. However, the general opinion is that in all probability the demand of the senescent or senile body does not exceed the requirement of a child. 400 to 800 I U (International Units) daily. It is entirely possible that it may be less than this amount.

Vitamin D deficiency in the aged may manifest itself as osteomalacia. There may be extensive osteoporosis and symptoms of bowing of the extremities and other bony deformities, pains in the ribs, sacrum and legs, emaciation and easy fatigue (see p. 694). The serum calcium and phosphorus are reduced and there occurs an increase in serum phosphatase. In treatment a vitamin D preparation containing 10,000 U S P units per gram may be given in doses of 15 drops three times a day. Larger doses should be used if the severity of the condition warrants it. The administration of calcium salts without vitamin D may intensify the symptoms.

VITAMIN D IN TREATMENT OF ARTHRITIS Enormous doses of vitamin D have been applied by some clinicians in the treatment of arthritis with some apparent success, but there exists a wide difference of opinion as to the benefits which may be expected from this form of therapy (see p. 681). An extensive review of the use of vitamin D is given by Reed, Struck, and Steck.¹⁸ The doses recommended in the treatment of arthritis are 150,000 to 200,000 U S P units daily. Doses up to 500,000 I U and even larger have been used. With doses of vitamin D of this magnitude, the danger of producing excessive systemic calcification must be kept in mind. The early symptoms of intolerance or excessive dosage are nausea, occasionally with vomiting, frequency of urination, lassitude, epigastric pain, and diarrhea. Vitamin D administration should be discontinued immediately when symptoms of intolerance appear, or serious damage may occur. Serum calcium determinations should be made before and during treatment, and elevation to a level above normal is indication for discontinuing treatment.

In view of the uncertainty of the benefit to be expected from this form of therapy of arthritis, it should be considered to be still an experimental form of treatment and thus applied only under careful control and with great caution.¹⁹ Cardiovascular and renal disease contraindicates the use of vitamin D in this range of dosage.

Vitamin K Deficiency The so called antihemorrhagic vitamin is of importance to the aged in liver or gallbladder disease where there is interference with the flow of bile to the intestinal tract. In such cases vitamin K, normally present in abundance, is not absorbed and the prothrombin defi-

of 4 to 12 mg. 2 methyl 1,4-prothrombin to normal in

human medicine, namely

pyridoxine, inositol, biotin, para-aminobenzoic acid, α -tocopherol (vitamin E), and pantothenic acid, is still too uncertain to make any definite statements as to their use or dosage.

Mineral Deficiencies. A large variety of minerals is necessary for the maintenance of the normal functions of the body. However, most of the minerals are required only in minute amounts and are obtained in sufficient quantity in the usual diet. Larger quantities of phosphorus are necessary but since it occurs in all natural foodstuffs, no special dietary considerations are necessary. In the light of present knowledge only two minerals appear to be of sufficient clinical importance to warrant special attention here. These are calcium and iron.

Calcium. The recommended allowance of calcium for an adult is 0.8 gm daily. Since the diet of the aged is likely to be deficient in calcium, particular care should be taken to see that the diet is constructed so that it contains sufficient calcium. If the patient shows evidence of demineralization, or in cases of fractures with delayed union, adequate supplies of both calcium and vitamin D should be assured. It should be remembered that excessive administration of calcium salts by mouth may produce nausea, vomiting, and diarrhea or constipation. When parathyroid hormone is being administered it is especially important to insure an adequate calcium supply in order to avoid demineralization. Serious consequences may follow continued abnormal elevation of serum calcium from administration of parathyroid hormone. Calcium therapy is contraindicated in osteitis fibrosa diffusa. Foods that are good sources of calcium are milk, cheese, ice cream, green vegetables and legumes.

Iron. The recommended allowance of iron for an adult is 12 mg daily. Intakes habitually below this level are very common. In order to avoid iron deficiency anemia, the daily diet should contain an adequate supply of iron. The elderly may require supplementary administration of iron salts. The administration of iron in anemia is discussed elsewhere (see p. 203). Foods that are good sources of iron are egg yolk, lean meat, liver, legumes, whole grain cereals, whole wheat or enriched bread and flour, molasses, dried apricots, dates, peaches, currants, and raisins.

DIETARY REQUIREMENTS OF THE AGED

In preparing diets for the aged it is especially important that an adequate vitamin intake be assured, since the caloric intake is likely to be low in order to prevent obesity and the food selection limited. It may be desirable to give special vitamin preparations to many of these patients. Since as yet no special differences in vitamin requirements in relation to age are known, it is safest to give the daily amounts recommended for an adult. The total daily intake for a normal adult should be as follows:

Vitamin A	5000 I U
Vitamin B ₁	1.8 mg
Vitamin C	75 mg
Riboflavin	2.7 mg
Niacin	18 mg

These quantities represent the amounts necessary daily for the maintenance of health in the average adult. The intake should be at or above this

level at all times. If evidence of deficiency disease is present, considerably larger amounts are necessary for treatment.

Rafsky and Newman's²⁰ observations indicate that vitamin supplementation in the aged may be necessary more frequently than was previously thought, even in apparently normal aged individuals.

Freeman²¹ feels that the diet for the aged should be high in protein, moderate in carbohydrates, low in fat and with higher than average vitamin levels.

If assimilation is normal and there is no need for special dietary restriction, vitamins as well as the other necessary nutritional essentials, can and should be obtained from the daily food supply.

The basic diet should be as follows:

- One pint of milk or its equivalent. Much of this may be utilized in creamed soups, puddings, milk toast and as cheese, etc.
- One serving of orange, grapefruit, tomatoes, or their juices. Raw cabbage or salad greens may be substituted if desired and no alimentary contraindications exist.
- One serving of green or yellow vegetables, some raw.
- One serving of potatoes or other vegetables or fruits.
- One serving of whole grain cereal—oatmeal or wheat.
- One egg daily, or at least three or four a week.
- One serving of meat, poultry, or sea food, or other high protein food.
- All bread and flour should be enriched or whole grain.
- Butter, oleomargarine with vitamin A, peanut butter, or other vitamin rich fats as indicated.

A diet constructed on this basis will meet all normal nutritional needs, if it is properly assimilated. The caloric content can be built up to any desired level by adding such other foods as may be desired.

If a fair trial of such a dietary regimen still leaves a question concerning nutritional well-being, there should be no hesitancy in the use of adequate vitamin supplementation as indicated by the individual case.

The prescribing of diets requires consideration of the duration of the dietetic program. During a transient, brief illness the diet may be grossly restricted without great detriment, but in advising anent diets to be used over a longer period of time, it is essential that the program include enough of the necessary vitamins and minerals. If it is desired to make a very restricted special diet in which the above listed basic foods cannot be used, it will be necessary to add special vitamin or mineral preparations.

It must be kept in mind that special methods of food preparation are frequently necessary for aged patients because of difficulty with mastication, digestive debility and deeply ingrained habits or prejudices. If the patient is edentulous, a soft diet is necessary or there will occur inadequate assimilation as well as colonic irritation. However, an adequate soft diet can be easily made from the same basic foods by properly pulping, straining, and grinding the necessary foods.

Consideration of the patient's long established habits, likes and dislikes, and personal dietetic convictions is necessary if the physician is to be successful in persuading the senescent or senile individual to eat properly. Imagination, meticulous attention to detail and compromise (which must not, however, allow any deficiency to arise) can spell the difference between successful dietetic control and total failure due to lack of cooperation.

REFERENCES

- [illegible]

CHAPTER 13

DISEASES OF THE BLOOD

RAPHIAEL ISAACS

HEALTHY individuals more than sixty years of age do not show any characteristic abnormalities of their blood. There is a tendency for the older person to show a slight average decrease in the red blood cell count and hemoglobin percentage, but it is not below the minimum normal for younger people. The average red blood cell count of 156 men, fifty years or older, was 4,600,000 per cu mm (range 4.0-6.1), and for women, 4,450,000 per cu mm (range 3.9-5.5). The hemoglobin average for men was 14.22 gm (range 13.1-16.5) and for women 14.15 gm (range 12.4-15.5) per 100 cc. Even after the menopause most women continue to have a slightly but definitely lower value in the number of red blood cells and hemoglobin content as compared with men of the same age, although because of the range of values, individual women may have a higher erythrocyte count than individual men. The leukocyte counts vary from 6,000 to 10,000 per cu mm, with no evident characteristic change in the cell types or numbers. Factors influencing the blood cell count are pulmonary emphysema (increase in the red blood cell count), arteriosclerosis, hypertension (with occasional hemorrhage), and achlorhydria (defective iron digestion).

It is rather curious that the blood cells should always be young and freshly formed, though many of the body cells are aged. Aging, however, is reflected in the ability of the bone marrow to produce cells in adequate numbers under conditions of strain or emergency. Thus recovery from

about the same in the aged
variation in the relative per-
centages from individual to individual. Normal blood formation has been reported up to the age of 135 years. The blood platelet number may be the same or slightly lower than in younger individuals.

The opsonic index of the serum and the phagocytic activity of the white blood cells have been reported to be diminished in amount. The sedimentation rate is accelerated, more markedly in aged women. The rate is more rapid in the second hour than in the first. The changes have been related to the degree of arteriosclerotic processes as well as to change in the albumin-globulin ratio of the serum. The glycolytic activity of the blood appears to be greatly diminished in individuals 95 to 138 years of age.

In some of the older patients there is a decrease in the total blood volume (See p 148.)

PERNICIOUS ANEMIA

Definition. Pernicious anemia is a chronic condition characterized by a macrocytic, hyperchromic anemia, neurologic (mostly sensory) and gastrointestinal symptoms, ease of fatigue, achlorhydria, and atrophic glossitis. The patients have a definite constitutional type.

Incidence. Pernicious anemia is not uncommon in the aged. The age incidence in a group of 500 patients was as follows:

Up to 29 years	2.5%
30-39 years	7.0%
40-49 years	21.5%
50-59 years	32.5%
60-69 years	29.0%
70— years	7.5%

In 1939 there were 1347 deaths in the United States (Metropolitan Life Insurance Company statistics) from pernicious anemia in patients sixty-five years and older (65-69 years, 341, 70-74, 392, 75 and over, 614). This is a rate of 15.4 per 100,000 population (60-69 years, 9.2, 70-74, 15.6, 75 and over, 24).

There appears to be no sex predisposition.

Etiologic Factors. The mechanism of anemia production is the deficiency in the megaloblast maturing substance. The neurologic changes appear to be sequelae of the same cause which produces the stomach defect, but are not related directly to the degree of anemia.

An enzyme-like substance produced in the stomach normally acts on some element of the food producing a material which, after storage in the liver, has the power of stimulating immature red blood cells (megaloblasts or proerythroblasts) to mature. This substance (liver extract) is apparently vitamin B₁₂. When it is deficient in amount, as in pernicious anemia, the maturation of the red blood cells is blocked and but few ripe forms develop.

Symptomatology. The symptoms in the aged are much like those of the younger groups. There is such a gradual development of weakness and ease of fatigue that it is difficult for the patient to give the exact date of onset. He notices dyspnea on slight exertion, and he may have anginoid precordial pains. There may be stubborn constipation, more rarely diarrhea. The tongue may become sore and atrophic, or there may be no visible erosion or other lesion (Fig. 42). The patient may notice some clumsiness in walking or handling objects, and there may be numbness and tingling of the feet and hands. Some notice that they have difficulty in buttoning their clothes. In the more advanced cases cystitis, with its characteristic symptoms, develops. Pain is not common, although muscle and joint pains may be symptoms which first bring the patient to the doctor. The pain may be constant or sharp and shooting in character. A common complaint is that of a band sensation, especially in the abdomen. Cerebral manifestations may include mild depression, indifference or apathy, irritability, delusions, hallucinations, disturbances in memory, violent maniacal outbursts, and coma. A change in personality is often noted and mistakenly attributed to "age."

retained and the pupillary reflexes are normal (difference from tabes) There may be unilateral or bilateral loss of position sense in the toes, ankles, or knees, with ataxia on locomotion and Rombergism Sensation is lost in areas of the skin with development of decubitus ulcers The loss of sensation in the gastrointestinal tract manifests itself in constipation, occasionally diarrhea, and in the bladder, by retention of urine and subsequent cystitis and pyelitis

In the aged, the *gastrointestinal symptoms* may be prominent. There is loss of appetite and occasionally the diet is remarkably limited. Nausea and vomiting are experienced at some time in the disease by about half of the patients. A feeling of heaviness after meals and an unusual tendency to "gas" accumulation may be troublesome symptoms. "Gallbladder attacks" with pain and tenderness in the right upper quadrant of the abdomen, and attacks of deepening jaundice with nausea, are more common in pernicious anemia patients than in the usual hospital population.

The heart is usually not enlarged, and there may be little anatomic evidence to explain the anginoid pain which may appear after exertion. The



Fig 42 Print of tongue of patient with pernicious anemia, on smoked paper. The areas with the intact papillae show the fine stippling. Sore and atrophic areas appear smoother.

electrocardiogram may show low voltage. Hemic murmurs, systolic in time, may be heard over the whole precordium. The pulse rate is usually increased in proportion to the anemia. The *blood pressure* may be lower than one would expect from the individual's age. The *spleen* is seldom palpable, but may be larger than normal. *Fever* may appear at times, is rarely high, and seldom presents a definite recurrent pattern.

Mental changes may show themselves by defective memory, indifference, disorientation, irritability, somnolence, melancholy or delirium

Diagnosis. The main diagnostic features are the hyperchromic macrocytic (oval red blood cell) anemia, leukopenia, achlorhydria, sore tongue, neurologic complications (sensory), elevated blood bilirubin, gastrointestinal symptoms, and tendency to the development of decubitus ulcers

Pernicious anemia may be suspected in the older individuals by the presence of a constant sense of fatigue, peculiar uniform pallor, numbness and tingling of the extremities, constipation or diarrhea, loss of walking because of loss of sensation, vibration sense in the legs, long narrow ears, and early graying of the hair.

Of these symptoms, achlorhydria is not uncommon in the aged. It may be expected as a "normal" finding in about 25 per cent and in higher percentages in the very old. Diminution of the vibration sense in the legs is more common in the normal aged person, hence is of greater diagnostic significance in younger patients. Cystitis is another symptom which may commonly arise from other causes in the aged.

The information derived from the blood is of great importance. While neurologic changes may precede the anemia and make early diagnosis quite difficult at times, the appearance of the red blood cell changes simplifies the problem. The first change is the appearance of a high color index. Another measure of this is a high hemoglobin-erythrocyte ratio (three times the number of grams of hemoglobin, divided by the number of hundred thousands of red blood cells. Normally the fraction is about equal to 1, in pernicious anemia the numerator is higher than the denominator, i. e., $1+$).

The degree of anemia may be mild (3,000,000 red blood cells per cu mm) or severe (700,000 per cu mm). There is a wide range in the diameter size of the red blood cells, from very small to very large (3 to $13+$ microns). The macrocytes tend to be oval, rather than round. The mean corpuscular volume is increased above 100 cubic microns. The platelets are reduced in number and leukopenia is present. The icterus index may be elevated to 10 or 20 or higher, with a blood bilirubin of 1.25 mg per 100 cc of blood, or higher.

Bone marrow, obtained by sternal puncture, shows an abnormal number of red blood cells in the megaloblast stage.

Differential Diagnosis In the aged, progressive anemia with pallor may be associated with chronic hemorrhage, sometimes secondary to cancer. In these there is iron deficiency anemia with small round red blood cells, and the icterus index (blood bilirubin) is not increased. Blood may be found in the stools. The presence of free hydrochloric acid in the stomach after histamine stimulation rules out pernicious anemia. In cirrhosis of the liver, hypothyroidism, intestinal stenosis or anastomoses, carcinoma of the stomach or head of the pancreas, or sprue, the red blood cells are large and round instead of oval, and microcytes are rare.

In tabes besides positive serologic data, the pupils may not react to light, the knee jerks are usually absent, and Achilles tendon tenderness is lost, the opposite conditions characterizing pernicious anemia.

Course In the untreated condition, the disease is progressive with relapses and remissions. Neurologic changes are progressive, ultimately crippling the patient. With adequate treatment the anemia disappears, usually within two months. The progress of the neurologic changes may be checked by intensive treatment, but many of the changes are irreversible. The feeling of weakness may persist for a long time, but most of the subjective symptoms improve rapidly with the elevation of the red blood cell count.

When the neurologic involvement is severe, decubitus ulcers may develop over the bony prominences. With loss of sensation in the bladder, there is urinary retention, occasionally with "overflow." Cystitis may develop and, ultimately, pyelitis (see p. 611).

Treatment The treatment of pernicious anemia consists primarily in giving enough *liver extract* to keep the red blood cell count normal. The initial dose should not be less than 1 U. S. P. unit a day, and preferably nearer

5 units a day during the period of severe anemia. When given by intramuscular injection, 10 to 15 units may be given at one time and repeated every two or three days. As the red blood cell count approaches normal the intervals may be lengthened until about 10 units are given per week. When infection is present, it is wiser to use higher doses. When the red blood cell count reaches 4.5 to 5 million per cu mm (usually in about six to eight weeks), a maintenance dose must be determined. This will usually be from 10 to 15 units a week to 15 units once every two or three weeks. Various oral preparations are available. The daily dose must be one or more units.

Folic acid, 20 to 50 mg daily by mouth, or 150 mg intramuscularly every two weeks, is valuable in producing a hematopoietic remission, but may not prevent the development of neurologic complications. (See p 288.)

As accessory medication, when there is iron deficiency, ferrous sulfate or carbonate 3 to 5 grains may be given three times a day. Dilute hydrochloric acid, 4 cc in a glass of water with meals, is necessary only when there is much discomfort after meals or diarrhea. Arsenic has been displaced by liver extract.

For the neurologic symptoms, *purposive exercises* are helpful. Each organ should be made to go through purposive movements, normal for that organ. For the fingers, tying knots in string and untying them, bead work, and similar exercises should be persisted in for weeks. Walking under controlled conditions with as much help and support as necessary, should be practiced several times daily. Occupational therapy is valuable. Preparations of vitamin B, especially B₁ and B₁₂, have been recommended.

The patient should be confined to bed as little as possible. The symptoms appear to progress with bed rest.

Reddened areas on the back should be painted daily with collodion solution. *Ulcers* should be washed clean and exposed to the light of an ordinary 25 or 50 watt electric light bulb at a distance of about 12 to 18 inches. When the patient must turn or when the light must be discontinued temporarily, a wet dressing with aluminum acetate solution or a saturated solution of boric acid should be applied. At night dressings with balsam of Peru in castor oil may be used. Acriflavine or merthiolate may be applied. The circulation around chronic ulcers may be stimulated by gentle massage or alternate suction and pressure applied with a rubber bulb attached to the stem of a funnel. Care must be used to remove feces from involuntary bowel movements and to keep the skin dry.

Where there is evidence that there is *urinary retention* (bladder distention, dribbling), pressure may be applied over the bladder (method of Credé) every three hours. The prognosis after catheterization is less hopeful. In severe cases, however, the bladder may be irrigated every two to four hours with 0.25 per cent acetic acid solution. Methenamine and mandelic acid preparations may be given by mouth, and sulfadiazine or others of the similar compounds may be used. Sometimes changing the reaction of the urine from acid to alkaline and vice versa, or giving large amounts of fluids, is helpful. Complete emptying of the bladder at regular intervals, however, is necessary (see p 642). Penicillin may be effective.

The soreness of the *tongue* usually improves after the return of the blood count to normal. Temporary relief may be obtained by touching the sore areas with a 1 per cent solution of silver nitrate.

When the *mental symptoms* are marked, restraint and careful watching are necessary

Constipation is difficult to combat, for cathartics which work by sensory stimulation are ineffective. Enemas of various types (soapsuds, glycerin, glucose) are frequently necessary, but they may not be effective. Surgical pituitrin, 0.5 to 1.0 cc., acting directly on the muscle, may be injected hypodermically once or twice a week.

For the *pain* in the legs, cramps and contractures, physiotherapy methods give the most hopeful results. These include heat, massage, and passive exercises. Acetylsalicylic acid, codeine, and other *sedatives* may give temporary relief. If traction apparatus is used, care must be taken to avoid skin injury.

There are no specific limitations in the *diet*, other than the ability of the individual to digest the different types of food. If liver extract is given regularly by intramuscular injection, it is not necessary to eat liver. It is wise to review the patient's diet at intervals to see if it is well balanced.

Complications Among the various complications encountered are oral sepsis, neoplasms of the stomach or prostate, cholecystitis or cholelithiasis, hemorrhage, arteriosclerosis, nephritis, paralysis agitans, edema, myxedema, diabetes mellitus, vitiligo, arthritis, and infection.

Prophylaxis As pernicious anemia is more common after the fifth decade patients who do not secrete hydrochloric acid in their stomachs should be watched for signs of anemia or for the neurologic changes. This is especially true in those who were prematurely gray. The presence of a high color index, even in the absence of gross anemia, is suggestive. The early use of liver extract may save the patient long periods of disability. Numbness and tingling of the extremities is often a warning sign.

It is important to prevent decubitus ulcers, cystitis, and contractures, and these complications should be kept in mind in the routine visits to the patients.

Prognosis The outlook for the control of the anemia is excellent if enough liver extract is used. A few individuals remain resistant, especially after the red blood cell count has reached the level of 3 million per cu. mm. In some of the older individuals who show an iron deficiency in the course of their treatment, iron is of value at this time.

Neurologic involvement is serious in the aged, and the prognosis for improvement is not good. We have seen individuals beyond seventy years who have regained the ability to walk, however, and whose decubitus ulcers have healed. These patients have had the benefit of intensive nursing and careful treatment. On the other hand, aged individuals confined to bed with but little nursing care soon lose their ability to walk, develop indolent ulcerations over their bony prominences, and cystitis and pyelitis terminate the picture.

With the advent of the use of liver preparations the outlook for pernicious anemia patients has improved greatly. In the Metropolitan Life Insurance Company's statistics the death rates for pernicious anemia per 100,000 population (white persons) for individuals forty-five years old and older, in the periods 1921-1925 and 1933-1937, were as follows:

1921-1925			1933-1937	
Age	M	F	M	F
45-54	6.4	10.1	2.5	2.9
55-64	14.3	21.4	6.3	7.5
65-74	18.2	27.5	15.0	17.2

HEMOLYTIC ANEMIA

Hemolytic anemia of the acquired type may appear in the aged. It may be idiopathic, or may be secondary to drugs, poisons, infection, hemolysins, or the effect or products of neoplasms. The sulfonamide compounds may produce a hemolytic type of anemia in some individuals, whereas in others the hemolytic process appears to be an allergic phenomenon. A hemolytic anemia may appear when there is faulty fat absorption, associated with a deficiency in the secretion of bile.

Symptoms. There is usually some degree of *jaundice*, often mild, and it may be evident only in the slight icterus of the sclera. The blood serum is yellower than normal (elevated icterus index) and the van den Bergh reaction is indirect. In prolonged hemolytic icterus, gallstones and obstructive jaundice may complicate the picture. The degree of bilirubinemia and its visible manifestations, as well as the presence of urobilinogen in the urine, are proportional to the ability of the liver to absorb and metabolize the products of abnormally rapid red blood cell destruction.

The essential features of the *blood* are the parallel reduction in the number of red blood cells and the hemoglobin content. The red blood cells are usually spherical (small diameter, normal or large mean corpuscular volume). The red blood cells may be large in some individuals, and, if hemorrhage is present, they are small and hypochromic. The bone marrow shows hyperplasia with a great increase in the number of normoblasts.

In the *differential diagnosis* in the older individuals, it is necessary to

acholic. In combinations of hemolytic and obstructive jaundice the spherical

Treatment. The cause of the hemolysis must be removed. Rarely is the operation having
 19 individuals No
 of Lederer, blood
 transfusion is helpful. In the secondary hemolytic anemias, blood transfusion may be followed by more rapid blood destruction, but it is frequently necessary to give blood when the red blood cell count is very low.

Some patients have improved when the intake of calcium is increased (calcium lactate, 15 grains [1 gram] three times a day). Vitamin A, 25,000

units two or three times a day, has been used, and some improvement has appeared to follow the use of pectin, 15 grains (1 gram) in capsules, three times a day one-half hour before meals

IRON DEFICIENCY ANEMIA

Definition The iron deficiency anemias include a group in which there is a relative defect in the amount of hemoglobin in the red blood cells, with a normal or decreased red blood cell count. The condition may result from acute or chronic hemorrhage, or from failure of the individual to ingest or absorb sufficient iron.

Incidence. Iron deficiency anemia is the most common type of anemia encountered in the aged. The most frequent abnormality in the blood of individuals more than fifty years of age is a low color index, or hemoglobin-red blood cell ratio. It was present in about 70 per cent of this group who were observed for various illnesses.

Etiologic Factors. While the loss of iron by men and by women after the menopause is quite small, metrorrhagia in the older group of women may be a serious factor (carcinoma of the uterus). *Cancer* itself does not lead to anemia as a rule, except when bleeding occurs. The most severe iron deficiency anemia is found when there is a carcinoma of the colon, especially the cecal end, and a color index of less than 0.5 is not uncommon. In bleeding gastric carcinoma the anemia may be severe, and in other tumors, in proportion to the blood lost.

A *defective food intake*, especially over long periods of time, may be followed by an iron deficiency. This is encountered in edentulous individuals, or those with chronic "dyspepsia" and "indigestion." These patients discard certain elements of their food because they cannot chew them or because they "disagree" with them. A diet may be reduced to crackers and milk or a similar simplified regime, which, after a longer or shorter period, is reflected in an iron deficiency. There may also be some difficulty in digesting the iron in the food and absorbing it. Achlorhydria and hypochlorhydria are more common in the aged than in the young, and this may be a factor in the lessened availability of food iron.

While the bone marrow may be qualitatively relatively normal, there may be some diminution in the amount, as a result of disease processes. Some individuals appear to be unable to use iron properly. In these patients inorganic iron salts are often ineffective in increasing the hemoglobin content of the red blood cells.

Symptomatology. Symptoms in iron deficiency anemia are proportional to the degree of myocardial insufficiency. If the heart muscle is strong enough to compensate for the lowered oxygen carrying power of the blood, there will be no symptoms. As soon as the heart becomes inadequate, dyspnea, ease of fatigue, edema, and all the other phenomena associated with inadequate myocardial

ness of the skin, its vascularity, and the amount of blood in the peripheral capillaries are factors entering into the skin color, and some of these may neutralize the effect of decreased hemoglobin content.

In the chronic forms there may be soreness of the tongue and the corners

of the mouth, and occasionally changes in the nails (koilonychia) may be present

Diagnosis A careful blood study is a necessary part of the examination of the aged. The iron deficiency may be judged from the lowered color index, lowered hemoglobin red blood cell ratio, lowered denseness of the stained red blood cells (more transparent than normal), normal icterus index or blood bilirubin content, and usually microcytosis. In anemias of chronic hemorrhage, elongated, pencil-shaped red blood cells may be noted in the film, the average cell size is small, and the leukocyte count and platelet numbers are normal or decreased. In acute hemorrhage, or in acute episodes during chronic hemorrhage, the platelet and leukocyte numbers increase.

Differential Diagnosis. The red blood cell measurements (diameter or volume) help to separate the macrocytic anemias from the microcytic anemia of hemorrhage. In the first stage of acute hemorrhage, the red blood cells may swell because of the hypotonic fluid replacing the lost plasma. In this condition the concentration of the hemoglobin is reduced in each red blood cell, and the difference between these and the well colored macrocytes of pernicious anemia, cirrhosis of the liver, or sprue is clear. In patients with cirrhosis of the liver and chronic hemorrhage a microcytic, hypochromic anemia may develop with the chronic blood loss.

Treatment While dietary iron is adequate for a normal individual, it is inadequate to supply the needs in iron deficiency anemia. Of the *therapeutic iron preparations*, the following may be used. Ferrous sulfate or carbonate, 3 to 5 grains, three times a day, reduced iron, 5 to 10 grains, three times a day. There is some question as to the best time to give the iron, before, with or after meals. It may require experimentation in each individual patient to note which time produces the best results. Usually enough copper is present in the food or in the iron preparation to supply the needs of the body. There appears to be some value in the use of whole liver in addition to the iron preparation. Liver extract (as for pernicious anemia) is of questionable value in iron deficiency anemia. There are many types of food and accessory food factors which are of value to the well individual, but which cannot be emphasized to their proper degree when the person is sick. One must depend on the iron medication and as near a normal diet as the patient can eat.

Bed rest is not necessary or wise if the patient can be up. Exercise must be limited to the amount permitted by the heart muscle. A simple test is to ask the patient to hold his breath as long as possible, without previously taking a deep breath. When the time is less than 15 seconds, activity must be very limited. Certain emotional or mechanical factors which interfere with this test should be recognized and evaluated.

Attention must be given to adequate *bowel movements*, as iron preparations have a constipating effect in some. The use of cathartics depends on the degree of health of the individual, and the underlying disease. If the patient is up and around, cascara preparations may be used. Iron and ammonium citrate, 4 cc of a 50 per cent aqueous solution may supply iron and have a loosening effect on the bowels at the same time. It is wise to inform the patient that his stools will be black or discolored after the iron medication.

Arsenic is usually not needed, and hydrochloric acid is necessary only

for individuals with achlorhydria who have a feeling of heaviness in the epigastrium after meals

Prophylaxis. In the routine check-up of older patients, iron deficiency, recognized from the blood count, may be corrected and the cause determined. When the lowered hemoglobin does not respond readily to medicine, suspicion of underlying trouble warrants further intensive study (see p. 115).

Prognosis. When the underlying cause can be removed or corrected, the prognosis is good, although the response may be slower than in young individuals. Patients with bleeding neoplasms, especially in the gastrointestinal tract, may show little or no response to iron by mouth. There may be some justification in these individuals for the use of intramuscularly injected preparations: iron and ammonium citrate, 1 grain daily or three times a week. The amount is limited by the reaction of the patient.

LEUKEMIA

Definition. Leukemia is a condition in which one of the types of leukocytes, or occasionally tissue cells, grow beyond the requirements of the body, the stimulus eventually becoming so great that the cells cannot mature. Acute or "blast" types and chronic forms are encountered. When the leukemic process is confined to the blood-forming organs, the disease is aleukemic or subleukemic, whereas the leukemic form is characterized by overflow of great numbers of immature forms into the peripheral circulation.

Incidence. The greatest incidence in chronic myelogenous leukemia occurs between the ages of twenty-five and forty-five, although cases have been observed in individuals seventy-five years old. After the age of forty-five the incidence is much less than in the younger individuals and patients more than sixty years of age are relatively uncommon. The majority of patients with chronic lymphatic leukemia are between forty-five and sixty years of age. The disease is relatively less common after sixty years. Monocytic leukemia has its greatest incidence in the decade between fifty-five and sixty-five years, 59 per cent of the patients being over fifty years.

The acute forms of leukemia occur most frequently early in life, in comparatively few patients beyond fifty years. In one series of twenty-seven cases occurring in those between the ages of sixty-one and seventy-five years, six were of acute, twelve of chronic lymphatic, and nine of chronic myelogenous leukemia.

Leukemia is more common in men than in women. In acute leukemia, in those between the ages of fifty and sixty years, the ratio is about 67 per cent males to 33 per cent females. In chronic lymphatic leukemia, between the ages of fifty and eighty years, the ratio is about 75 per cent males to 25 per cent females. In chronic myelogenous leukemia, between the ages of fifty and eighty-five, the ratio is about 56 per cent males to 44 per cent females. In monocytic leukemia, in corresponding age groups, the ratio is about 70 per cent males to 30 per cent females.

In 1939, in the United States, there were 966 deaths from leukemia in individuals sixty-five years and older (65-68 years, 363, 70-74 years, 290, 75 years and older, 313). Per 100,000 population, this is 11.0 for the ages of sixty-five and over (9.8 for ages 65-69 years, 11.6 for ages 70-74 years, 12.3 for individuals 75 years of age and over).

The death rate from leukemia appears to be increasing, although improvement in diagnostic accuracy may be an important factor. The following table shows the death rates per 100,000 population in the United States during 1921-25 and 1933-37 age groups

Age groups	1921-1925		1933-1937	
	M	F	M	F
55-64 years	3.9	2.4	7.1	5.1
65-74 years	4.2	1.8	10.1	7.1

Symptomatology. In the *chronic forms*, ease of fatigue is the first symptom. Later symptoms are those of a high metabolic rate: loss of weight, profuse perspiration, sensitivity to heat, irritability, nervousness, and insomnia. As anemia develops, there are, in addition, the symptoms associated with *circulatory insufficiency*. With enlargement of the lymph nodes, liver, and spleen, pressure symptoms are noted. Pain, cough, and gastrointestinal symptoms are sequelae. Cutaneous lesions and hemorrhages are late symptoms. Fever is common.

The *acute leukemias* may be discovered while investigating pharyngitis, pharyngeal and mucous membrane ulcers, and bleeding. There is usually a high fever when sepsis is prominent. There may be vaginal or rectal ulcers. Marked prostration comes early. Sepsis, following the extraction of teeth, is the presenting symptom in some cases.

The *leukocyte count*, in the chronic forms, is usually elevated, and immature cells, which crowd the marrow, also enter the blood stream. With the progress of the disease the cells appear more and more immature until the terminal blast or stem cell stage is reached. In the acute forms this is the state noted early in the disease. In the aleukemic forms the leukocyte count may not be elevated, but immature cells are present in the blood stream. There is usually a progressive anemia, running parallel to the degree of involvement of the bone marrow by the leukemic process and serving as a measure of the extent of the disease. Bone marrow puncture is a useful procedure when the diagnosis is obscure.

Treatment. Therapeutic agents in leukemia include x-ray, radium, radioactivated substances, Fowler's solution, urethane, and blood transfusion.

The choice of the best agency at any given time depends on the symptoms and the type of cells in the blood.

The response to *x-ray or radioactive substances* is good when the bulk of the cells are fairly mature (metamyelocytes or older, small lymphocytes). When many of the cells are immature (5 per cent or more of blasts) the results are poor and the prognosis after irradiation is worse.

X-ray or radioactive substances should be used *only* when there is overwhelming evidence of bone marrow crowding (progressive anemia) or when

the metabolic rate is marked. A high leukocyte count, loss of weight, profuse perspiration, sleeplessness, irritability, and tachycardia

would be indications for treatment. It must be remembered that x-ray and radioactive substances are not curative, but give symptomatic relief by reducing the leukocyte count and the basal metabolic rate.

Fowler's solution may be used in chronic myelogenous or chronic monocytic leukemia. The dose is started at 3 minims, three times a day, in water, after meals, and increased to within the tolerance of the patient. A dose of 3 cc a day may be reached in some patients although 7 minims three times a day is usually effective. Symptoms of poisoning may be herpes zoster, evidence of hepatic cirrhosis, keratosis, ascites, conjunctival and nasal congestion, diarrhea, edema of the eyelids and face, loss of appetite, vomiting, and joint and other pains. These symptoms require reduction of dose or temporary cessation of medication. Both x-ray therapy and Fowler's solution therapy are followed by a deepening of the pigmentation of the skin.



Fig 43. Leukemia cutis (monocytic) in a man aged sixty four years. Death followed within three weeks after the appearance of this symptom.

Penicillin, streptomycin, and sulfadiazine have been very useful in combating the infection in the disease, especially in the acute forms, or when the blasts predominate in the chronic forms.

Urethane in doses of 1 gram three times a day about one hour after meals may be very effective in reducing the leukocyte count. If there are temporary symptoms of intolerance (nausea, gastrointestinal disturbances, feeling of exhaustion or depression) the dose may be reduced. Aminopterin may be used, with caution, when the blasts predominate.

Blood transfusion may give considerable symptomatic relief in both the acute and chronic types of leukemia. It may be given every other day for three to five times in acute leukemia, or at monthly intervals in the chronic types, using the red blood cell count as a guide. The individual transfusion may be 250 to 500 cc, or 500 cc may be given on alternate days.

Liver extract is not effective in leukemia, but whole liver, about two to

three pounds a week, is of distinct value in the chronic lymphatic type. Iron or iron and copper may be given after a course of x ray treatments, or after the leukocyte count has been reduced by Fowler's solution, or during urethane therapy.

Prophylaxis Little is known of the predisposing causes of leukemia. It may be well for individuals chronically exposed to benzene to have blood studies made at intervals. Older individuals with unusual cells in their blood or with chronic leukopenia or leukocytosis should also be watched with leukemia in mind.

Prognosis. At present, the prognosis is very poor in acute leukemia. The duration may be from two to ten weeks after the first symptoms are discovered. The free use of blood transfusion prolongs the course. In the chronic forms, the average duration is about three and a half years, with some individuals living for twenty years or more. X ray treatment prolongs the life of individual patients and no doubt shortens the life of others, the average duration being much like that of the untreated cases.

POLYCYTHEMIA VERA RUBRA ERYTHREMIA

Polycythemia rubra is a condition in which there is an overproduction of red blood cells beyond the needs of the body. It is associated with defective oxidation in the bone marrow (thickening of the blood vessel walls).

The condition appears most commonly after the age of forty or forty-five years and may be noted in individuals older than seventy years. Males are more frequently affected than females.

Symptoms The disease begins insidiously, with increasing ease of fatigue. The patient becomes irritable, notices headache, joint pains, a throbbing sensation in the head, fingers or toes, tinnitus, dizziness and injection of the sclera. The color of the face is usually a crimson red (erythrosis) although rare individuals may not be abnormally red. Slight trauma leads to local thrombosis and ulcers may develop. There may be marked neurologic and mental symptoms.

Study of the blood shows a *red blood cell count* persistently over 6.5 to 7.0 millions per cu. mm., most commonly 8 to 10 millions. There is a definite *leukocytosis* and increase in the number of blood platelets. In spite of the high red blood cell count, immature forms may appear in the blood stream, reflecting the greatly heightened activity in the bone marrow. The total blood volume is increased. The red blood cells are small and the monocytes are reduced in number.

On *physical examination* the outstanding features are the erythrosis and the large spleen and occasionally an enlarged liver. In one group, the *blood pressure* is elevated (Geisböck's disease). It may appear between the ages of fifty or sixty years. *Albuminuria* is common.

Treatment The red blood cell count must be lowered. This may be

has been proposed, but this may be quite difficult to carry out in the aged. Prolonged remissions follow the use of radioactive phosphorus by

mouth or intravenously. A number of patients receiving this treatment have developed leukemia.

Complications. Cerebral hemorrhage, gastrointestinal hemorrhage, tendency to bleed after extraction of a tooth, perforation of gastrointestinal ulcers, leg ulcers, migrating phlebitis and thrombophlebitis, and necrotic lesions of the toes and feet comprise the complications. In the aged the elevated metabolic rate may show itself in the exaggeration of symptoms and in emotional instability.

Prognosis. The prognosis is good for from three to eight years in the carefully treated patients. Death may come from cerebral hemorrhage, thrombosis or infection. Rarely the disease terminates in anemia or leukemia.

SYMPTOMATIC POLYCYTHEMIA RUBRA ERYTHROCYTOSIS

The most common condition in the aged in which polyglobulism develops is pulmonary emphysema. The elevation of red blood cell count may be noted in some types of hepatic cirrhosis, pulmonary fibrosis, pulmonary and cardiac lesions, in which there is a chronic difficulty in oxygen absorption, and chronic or acute poisoning with certain drugs (benzene derivatives), arsenic, and phosphorus. It appears at high altitudes, in acute and chronic diarrhea of certain types, in thrombosis of the splenic vein, in pituitary basophilism, and in tuberculosis of the spleen.

In symptomatic polycythemia the spleen is not enlarged (unless the primary disease is associated with splenomegaly) and leukocytosis is not common. The red blood cell count may be elevated to 8,000,000 to 12,000,000 per cu mm. Hemorrhage and other symptoms of polycythemia may be present.

Treatment. This is directed to the underlying condition. The patient may feel worse when attempts are made to reduce the red blood cell count. Potassium iodide, 5 to 10 grains three times a day, may give some symptomatic relief. If the blood count must be reduced, venesection is the preferred method.

HEMORRHAGIC CONDITIONS

Etiology. A tendency to bleed, in the aged, may be present from many causes. These include platelet deficiency (thrombocytopenic purpura, leukemia, aplastic anemia from various causes, crowding lesions of the marrow, e. g., neoplastic metastases, pernicious anemia), capillary fragility (allergic purpura, vitamin C deficiency, vitamin K deficiency, toxic purpura) and chemical abnormality of constituents necessary for blood clotting (hypoprothrombinemia, especially in liver disease, hemophilia). In infections, platelet deficiency or the capillary fragility element may be factors. Some types do not fall inside these groups: familial tendency to bleed, hereditary thrombasthenia, hereditary hemorrhagic telangiectasia, bleeding associated with high blood pressure and polycythemia rubra.

Hemophilia is rarely a factor in old age. Primary hemorrhagic diseases were the cause of death in 47 individuals more than sixty years of age in the statistics of the United States, 1920. Five of these were 100 years of age.

Differential Diagnosis:

some

(other blood elements normal, except for results of hemorrhage, tourniquet test for capillary fragility, positive, clot retraction prolonged, bleeding time

prolonged, clotting time normal) or *leukemia* (immature leukocytes, leukocytosis, blasts) or *aplastic anemia* (all bone marrow elements reduced in number, few immature forms) In these conditions, bone marrow puncture gives positive data—increased number of megakaryocytes in thrombocytopenic purpura, increased number of blasts and mitotic figures in leukemia, gross reduction in cell growth in aplastic anemia

In the *nonthrombocytopenic purpuras*, the most common blood change is a mild eosinophilia (may be marked or negligible in individual cases), and the clotting and bleeding time and the clot retraction are not abnormal Capillary fragility is usually abnormal

Symptoms. Symptoms vary from pinpoint hemorrhages in the skin, mucous membranes, and retina, to extensive hemorrhages under the skin or into the internal organs The tendency to joint involvement is not so marked as in hemophilia Relapses and remissions are a common feature

Treatment. The cause of the bleeding will determine treatment In the *thrombocytopenic types*, blood transfusion is of symptomatic value Splenectomy, curative of the purpura hemorrhagica thrombopenica in younger individuals, must be undertaken less readily in older individuals Injections of whole blood or plasma, 50 cc at frequent intervals, intramuscularly, may control the bleeding Locally coagulen or Russell's viper or fer de lance snake venom may be used Pectin, orally in doses of three 5-grain capsules one half hour before meals, has been found to be very effective in controlling hemorrhage

In the *nonthrombocytopenic types* correction of the nutritional deficiency is required—ascorbic acid or vitamin K preparations, as the condition indicates Whenever possible the inciting cause must be removed (infection, drugs, allergy) When the capillary fragility persists, a course of injections of moccasin snake venom may be given In polycythemia rubra and in chronic myelogenous leukemia, reduction in the number of blood cells may lead to adequate blood clotting

Prognosis. When the cause is known and the defect can be corrected, the prognosis is good Cerebral hemorrhage or severe gastrointestinal bleeding may lead to death At autopsy, generalized punctate hemorrhages may be found in almost all of the organs The problem of operative measures, when necessary, is usually met by using multiple blood transfusions before and after surgical procedures

NEUTROPENIA MALIGNANT NEUTROPENIA* AGRANULOCYTOSIS

A decrease in the number of polymorphonuclear neutrophils may result, in susceptible individuals, from exposure to certain drugs (sulfonamides, amidopyrine, benzene and its derivatives, arsphenamine, thiouracil, dinitrophenol, gold salts, arsenic and radioactive substances)

Symptoms. The condition is characterized by the selective reduction in the number of neutrophilic leukocytes in the peripheral circulation, and the inhibition of their maturation in the bone marrow

Many diseases are characterized by too few neutrophils, associated with a relative increase in eosinophils and lymphocytes. The so-called *neutropenic* conditions, of which the most common is agranulocytosis, are usually associated with drugs, and, more rarely, in pernicious anemia, certain infections and septic conditions

When there are too few neutrophils, the tissues become invaded by bacteria and ulcers form in the mouth, pharynx, rectum, anus, and vagina. The patient becomes "septic" with fever, chills, and marked prostration.

Treatment. For treatment, one may give small blood transfusions, pentnucleotide, 20 to 40 cc daily, crude liver extract intramuscularly, and ascorbic acid, 100 mg daily, intravenously. The prognosis is serious until the patient responds to one or the other of the methods of treatment. The mortality rate has been reduced from about 80 per cent to 10 or 20 per cent in the type in which there is selective reduction in the number of neutrophils. In the other types the response to treatment depends on the underlying condition.

Penicillin may be of value in combating infection. Pyridoxine, by mouth or intravenously, has been used.

There are many individuals who have a *chronic neutropenia*, without evidence of disease.

MACROCYTIC ANEMIA

Large, well colored blood cells characterize any disease in which too little "liver extract" is available for maturation of megaloblasts in the bone marrow. The interruption in the process of manufacture, storage, transformation, and delivery of the "liver extract" is determined by the nature of the disease. Thus the gastric enzyme, hemopoietin, may be deficient in amount (pernicious anemia, gastric carcinoma, gastric resection) or the food to be digested may be deficient (vomiting, some cases of pellagra) or it may not be absorbed (sprue, intestinal anastomoses, intestinal stricture, ileitis, prolonged diarrhea, ulcerative colitis, types of dysentery) or the storage mechanism may be deficient (cirrhosis of the liver, hepatitis, hemochromatosis). Additional conditions in which one or more factors may play a part are myxedema, tapeworm anemia, and achrestic anemia.

Symptoms. The symptoms resemble those of iron deficiency anemia. The red blood cells are large in volume and diameter, well filled with hemoglobin, differing from those of pregnancy or acute hemorrhage, where the cells have become large by swelling, and thus show a lowered mean corpuscular hemoglobin concentration. The red blood cells differ from those of pernicious anemia in that they are predominantly round, instead of oval, and in that the shift in size is toward the large cells, rather than the wide range (very small and very large) found in pernicious anemia.

Treatment. For treatment, liver extract should be given by intramuscular injection daily, 2 to 5 units in the beginning, and more or less as a maintenance dose until the cause is remedied and danger of relapse is past. Occasionally a patient may not respond to liver extract intramuscularly, but will improve with a special liver extract intravenously. Blood transfusions may be necessary in those individuals who do not respond to the injections. It may be well to try yeast or yeast extracts (Vegex, 15 to 30 gm daily).

Folic acid, in doses of 20 to 50 mg daily by mouth or by intramuscular injection, may be very effective in some types of macrocytic anemia.

APLASTIC ANEMIA

This condition is characterized by an anemia in which all bone marrow cells in the blood stream are decreased in number (red blood cells, neutro

phils, monocytes, and platelets) The picture in the peripheral blood may appear when the marrow is aplastic and replaced by fat or fibrous tissue (osteosclerosis, myelosclerosis) but it sometimes is found even when the marrow seems to be quite cellular Aplasia of the marrow may result from idiosyncrasy to certain drugs, or benzene, arsenic, intense radiation (x-ray, radioactive substances) or as a terminal stage of conditions in which there has been a period of intense overgrowth or stimulation At times the cause is not evident (idiopathic aplastic anemia)

There is usually a severe anemia and leukopenia with reduction in the number of lymphocytes as well as neutrophils and blood platelets The icterus index is low, the color index normal, red blood cell size, normal, immature red blood cells absent in the peripheral blood

Symptoms. Symptomatically the disease is characterized by ease of fatigue, uniform pallor, tendency to bleed, and all the symptoms which go with taxation of heart muscle Ulceration may appear about the mouth or rectum, and a mild fever may be present

Treatment. The treatment consists in giving blood transfusions, 500 cc at daily to weekly intervals until the red blood cell count is around 4 or 5 millions per cu mm It is usually necessary to give 500 cc every two to four weeks when the regeneration of the red blood cells is at a very low level Among the drugs which may be tried are epinephrine (0.3-0.7 cc hypodermically, daily), thyroid, androgenic substances, and bone marrow infusions with marrow suspensions in blood Liver, iron, arsenic, and pent-nucleotide are ineffective in aplastic anemia

Prognosis. The outlook is usually bad, although a patient may be kept alive for one or two years with repeated transfusions Occasionally after months of transfusions, a patient may start to regenerate his marrow and recover

Prophylaxis Individuals receiving or those who are exposed to benzene or its derivatives, x-ray, radium, radioactive substances, lead, mercury, bismuth, arsenic, arsphenamine, gold salts, and sulfonamides should be watched for evidence of sensitivity A fall in the number of any of the bone marrow elements may be significant

OTHER DISEASES

While *sickle cell anemia* and *hemophilia* are encountered most commonly in the young, occasionally an older person presents the problems of these diseases The condition is usually milder, for the more severe forms cause death early in life A fracture of the head of the femur in a sixty-year old woman brought to light a case of *Gaucher's disease* *Erythroblastosis in*

viduals of the older groups

A hypoplastic type of anemia may accompany nephropathies, unless there is much blood loss In chronic infection there is usually an iron deficiency (hypochromic) type of anemia

CHAPTER 14

DIABETES MELLITUS

EDWARD L. BORTZ

GENERAL CONSIDERATIONS

Incidence and Mortality. Diabetes mellitus is steadily increasing in frequency in individuals over sixty years of age. Earlier diagnosis, coupled with vastly improved measures in treating diabetes, together with the general aging of the population, is the principal factor bringing about an increase in the incidence of the disease.

In the decade past, the mortality rate from diabetes in the group under forty-five years of age has gradually decreased, while it has steadily increased in the age group of sixty-five years or more. From data obtained in the National Health Survey of the United States Public Health Service, diabetes will probably increase approximately 20 per cent within the next ten years. The death rate from diabetes in 1950 is calculated to be approximately 25 per cent above the present rate and practically double that of a decade ago. However, if the aging of the population is taken into account the death rate in 1950 will be only 7 per cent above the present rate and about one-quarter higher than that of 1930.

Life Expectancy and Diabetes Mellitus. Since the turn of the century, spectacular advances in the field of medical science, together with improved social conditions, have brought about a striking extension in the life expectancy of the nation's population. In addition to these benefits, in which the nation's diabetic population participates, the discovery of insulin has furnished diabetics with an effective means for controlling their disease.

It is true that large numbers of individuals with diabetes succumb before they attain the later years of life. However, knowledge available concerning the nature of diabetes and methods for treatment is of such practicable value that for every diabetic who dies someone is probably to blame. The doctor himself may be at fault in his lack of understanding of proper diet regulation, the use of exercise, and the prescription of insulin. The modern treatment of diabetes today offers highly effective control measures to stay the ravages that occur within the body in the uncontrolled diabetic condition. Or it may be the patient who is at fault in failing to follow the physician's instruction, or, as too often happens, by sheer neglect and disinterest in his own welfare. It was Montaigne who once said "people don't die, they kill themselves."

Diabetes is most likely to appear between the ages of fifty and sixty

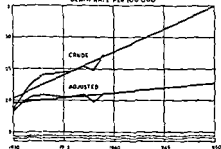
of fifty and one
between

f deaths occurred

MORTALITY FROM DIABETES

GENERAL TREND

DEATH RATE PER 100,000

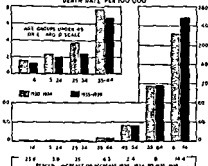


Data charts based on Mortality Experience of Metropolitan Life Insurance Company from the Department

The crude death rate from diabetes will increase rapidly because of the growing proportion of older persons, especially women in contrast to the slower rise in the sex age adjusted mortality

RECENT AGE CHANGES

DEATH RATE PER 100,000

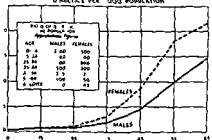


Diabetics die older now. The chief problem of the new decade is the better control of complications in the middle aged and older diabetics

PREVALENCE OF DIABETES

SEX-AGE LEVELS

DIABETICS PER 100,000 POPULATION



National Health Survey 1951-1952

A nation-wide survey shows the rapid increase in the known frequency of diabetes with advancing age. The actual prevalence is probably higher

TREND

INCREASES DUE TO OUR AGING POPULATION

ESTIMATED NUMBER OF DIABETICS AS PERCENT OF NUMBER IN 1930



Diabetes incidence in National Health Survey applied to present age, sex, and race of 1930 Census and the total population Census for 1940 and 1950

The trend of...

proved longevity of diabetics will raise the number even more. By 1950 diabetics in the United States may number one million.

Fig. 44 Trends of diabetes mellitus (George Baker Clinic of Boston and Metropolitan Life Insurance Co.)

With improved care, the life expectancy of diabetic patients has increased more rapidly than that of the general population, until now it is just as good as the anticipated survival of the nondiabetic population. From these facts, the brilliant investigator, Francis D. W. Lukens, of the University of Pennsyl-

vania, who has added so much to the modern understanding of diabetes, concludes "diabetes is not a complication of old age, rather, old age is a complication of diabetes, which has its peak of incidence before the sixtieth year" Lukens further states that "diabetes is a good disease with bad com

TABLE 4

Age	Male		Female		Total	
Years	Patients	Per cent	Patients	Per cent	Patients	Per cent
0-9	15	1.8	27	4.2	42	2.8
10-19	35	4.1	28	4.4	63	4.2
20-29	49	5.7	44	6.9	93	6.2
30-39	101	11.8	65	10.2	166	11.1
40-49	240	28.1	160	25.1	400	26.8
* 50-59	230	27.0	190	29.8	420	28.2
60-69	136	16.0	98	15.4	234	15.7
70-79	39	4.6	22	3.5	61	4.1
80-89	6	0.7	1	0.2	7	0.5
not stated	2	0.2	2	0.2	4	0.3
Total	853	100	637	100	1490	100
Mean	48.3		46.8		47.6	

This table is the latest report of Diabetes Mellitus (1940) Age at Onset by Sex by Edward H. Ryncarson, M.D., Mayo Clinic, Rochester, Minn.

panions." He points out that in the aged, diabetes is, or should be, at its best, and the patient and the doctor should strive to avoid those bad companions, the complications.

As many of these complications are due to arteriosclerosis, rather than to the diabetes *per se*, the control of blood cholesterol would seem particu

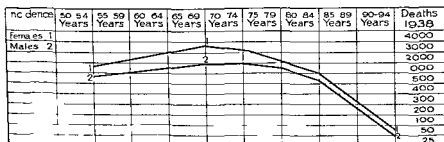


Fig. 45 Age of death from diabetes mellitus (From Vital Statistics Special Report 9)

larly desirable in the aged patient. In experimental animals, Lukens found that recovery of hydropic degeneration of the islands sometimes occurred in the presence of gross scarring of the pancreas. This suggests that the fibrosis of old age would not prevent the beneficial effect of treatment. The difficulty,

however, of making an early diagnosis renders any duplication of animal work most unlikely

Richter and Schmidt² show that their diabetic rats exhibit much less spontaneous activity than do normal animals. From this reaction they suggest that the meaning of exercise in the treatment of diabetes is not yet fully understood. Lukens suggests that this observation may have significance for the elderly diabetic who cannot tolerate exercise. It should provide consolation to the aged diabetic and stimulate his observation.

CHARACTERISTICS OF DIABETES IN THE AGED

Ordinarily, diabetes occurring after the age of sixty is apt to be slow and insidious in onset and mild in degree. The elderly patient is not nearly so likely to develop diabetic coma as is the youngster or young adult. However, every clinician who has cared for diabetic patients knows that advanced ketosis is possible with overindulgence in food and/or an undersupply of insulin, especially in the presence of infection. Usually the problems of diabetes in the elderly patients are those ordinarily associated with other complications rather than the development of acidosis. Elderly patients seem to have the ability to withstand more metabolic abuse than younger bodies. Signs of metabolic decompensation appear more slowly and likewise the action of insulin is apt to be less prompt in manifesting its control of the sugar curve. As Lukens states, "the aged are commonly less sensitive to insulin and have the widest margin of safety between control of the disease and reaction." Obese elderly patients frequently have the opportunity, which is usually denied to children, of improving the diabetic condition by some reduction in the body weight. Treatment of the diabetic in the later years should be instituted gradually and sudden changes in the regime should be avoided.

Clinical Types. Ryneerson,³ of the Mayo Foundation, divides his cases into four classes: (1) those controlled by simple qualitative restriction in foods rich in carbohydrates, (2) those patients who require a weighed diet, but no insulin, (3) those patients requiring both insulin and a weighed diet, (4) those patients that need more than 30 units of insulin daily.

It must be remembered, however, that while, as a rule, diabetes is of a mild degree in the aged, this does not exclude the possibility of a severe form occurring in the senescent years. Diabetes may remain latent for a long period of time and be brought to the surface by the development of a cold, by a severe infection of the skin, by the development of a gastrointestinal disturbance, or by any disease elsewhere in the body.

SYMPTOMS

Oftimes diabetes may not be suspected until the individual's body is placed under a strain, at which time sugar will be found in the urine and the subsequent analysis of the blood shows an increase in the level of the sugar. Not rarely, vision defects, arthritic pains, or loss of weight send the patient to the physician, and as a rule he gives a history of having an exceptionally good appetite and often may be moderately or greatly overweight. The history of weakness and sudden loss of weight may be elicited. Elderly people, particularly women, are frequently and thoroughly annoyed by

perineal pruritus This is due, of course, to sugar in the urine irritating sensitive tissues

Polyuria, polyphagia, and polydipsia, together with a tendency towards drowsiness, are frequent Occasionally the patient complains of loss of memory, and, in the unusual case, may show some psychic disturbance These symptoms, however, are not due strictly and solely to the diabetic condition, but their presence is due in a great degree to vascular degenerative changes (see p 260) Dermatitis is a frequent accompaniment of metabolic deterioration in the elderly (see p 737)

DIAGNOSIS

The very first suspicion that a patient's metabolism is not operating at optimum efficiency may be suggested by the presence of a trace of sugar in the urine Periodic health examinations and periodic urinalysis recommended by life insurance companies have rendered a great service by bringing early cases under medical management

It is necessary to make a thorough study of the blood sugar in order to complete the diagnosis of diabetes mellitus. *In borderline cases the glucose tolerance test should always be performed* When an increase in the blood sugar has been established, ~~not~~ only should the pancreatic inadequacy be held responsible, but a further search for other causes should be made Persons addicted to the excessive use of alcohol frequently show a diminished tolerance for carbohydrates Disturbances of the biliary tract or glands of the endocrine system other than the pancreas and intracranial lesions may likewise be accompanied by a high blood sugar The blood cholesterol is frequently elevated

A family history of diabetes further aids the diagnosis Little difficulty is experienced in making this diagnosis, once its presence is suspected Diagnosis must, however, include more than merely identifying the disease It must consider the *quantitative* aspects of the severity of the impairment

Nondiabetic Hyperglycemia An increase in the level of the blood sugar, transitory in nature, frequently manifests itself in the presence of certain other conditions The most common of these are (1) glandular disorders such as exophthalmic goiter, hyperthyroidism and hyperpituitarism, (2) hypertension, (3) arteriosclerosis, (4) nephritis, (5) hepatic disorders (6) carcinoma, (7) syphilis, (8) tuberculosis, (9) arthritis (10) any infectious or toxic state, (11) postanesthetic depression, (12) in normal subjects over fifty years of age

Diabetes may be coexistent with these other disturbances Furthermore it may be in the latent stage and be activated by the appearance of tuberculosis, nephritis, syphilis or carcinoma In any event the patient's blood sugar should be checked at frequent intervals and due precautions taken to minimize the strain on the metabolic function ⁴

Glycosuria without Hyperglycemia. Many patients are discovered to have diabetes by a routine examination of the urine associated with their application for life insurance When sugar is found to be present in the urine, the resourceful physician will not fail to have a thorough examination of the patient's blood Sugar will normally spill over, when the renal threshold is low, after a heavy carbohydrate meal It is possible that there may be some depression of the renal threshold for sugar so that glucose may escape through

the renal parenchyma at occasional intervals. Despite the absence of true diabetes mellitus, nevertheless the patient with glycosuria should be regarded as a diabetic until proven otherwise.

Case example A patient, a young doctor age twenty six, had shown sugar in the urine for three years, although during this entire time his blood sugar was not elevated. With a moderate limitation of diet and exercise, the sugar in the urine cleared up. Nevertheless, two years after the low renal threshold was identified, even with the normal blood sugar, the patient developed diabetes. For adequate control of his condition insulin was required.

Hyperglycemia in the Absence of Glycosuria. A high index of suspicion is a helpful rule to follow in discovering patients with diabetes. Unless a thorough examination of the blood is made, the metabolic deficiency may be entirely missed. The absence of sugar in the urine by no means rules out the possibility of the presence of diabetes, since many individuals have a high renal threshold and some carry a blood sugar of 400 mg per 100 cc without revealing sugar in the urine. At least occasional blood sugar analysis must be performed if the doctor supervising the patient is to have the information necessary to prescribe intelligently for the patient's care.

PROGNOSIS⁶

Discovery of the presence of diabetes in an elderly individual may prove a blessing to the patient. In the absence of other complicating factors, such as lesions in the heart, kidney, liver, or intestinal tract, the prescription of a well-balanced and varied diet, plus moderate exercise and adequate insulin, is followed by the prompt disappearance of the symptoms which disturb the patient. Increasing strength and well being are frequently noteworthy. Reasonable attention to sound tenets of health will guarantee against the onset of ketosis.

The elderly diabetic may do much for himself to conserve his waning energy and power. In addition to diet prescription, adequate elimination with attention to the bowels, kidneys, and skin, plus sufficient rest, with a midday siesta, will enhance the diabetic's chances for living his allotted life span.

Increasing evidence has been accumulated to suggest that complete control of metabolic inadequacy will at least retard the progressive development of arteriosclerotic degeneration of the cardiovascular system. However, the final answer to this problem still lies in the future.

In the light of modern therapy the elderly diabetic has reason for gratitude in the increase in years that he is facing. With good fortune he may live longer with the disease than would have been his lot without it.

SURGERY AND THE ELDERLY DIABETIC

Surgical procedures on a patient with uncontrolled diabetes are fraught with great danger. Tissue resistance is greatly reduced and this invites bacterial infection. To the subclinical acidosis, due to the incomplete fat metabolism, there may be added a superimposed acidosis from the anesthesia. It may happen that the first suggestion that the patient is diabetic may arise from a preoperative examination, which reveals an elevated blood sugar.

Control of Diabetes. Before patients are operated on the diabetic condition should be brought under control. Enough glucose and insulin should

be given to support the metabolism. A daily fluid intake of 3000 to 6000 cc should be insisted upon, together with generous quantities of sodium chloride and vitamin B. The urinary output should be over 1000 cc per twenty four hours. In the presence of a defective cardiac action, sodium chloride should be greatly restricted. Moderate limitation of fluid must then be considered.

The elderly diabetic who has been properly controlled carries the shock and strain of surgical procedures surprisingly well. In fact, he should carry it as well as the nondiabetic elderly patient. (See Chapter 8.)

To successfully meet the operation an elderly patient must have adequate stores of carbohydrates in the three major reservoirs of the body—the liver, the muscles, and the skin. It is very important that the blood sugar be reasonably near normal. However, the level in elderly patients may be kept safely from 140 to 200 mg, in order to avoid sudden drops in the blood sugar and the possibility of insulin reaction. The diet can be liberalized since insulin is available.

For major operations, where patients are not permitted or able to take sustenance by mouth, it is perfectly easy to administer glucose by the intravenous route, care being taken to cover the patient's deficiency by administering insulin hypodermically. Protamine insulin, with or without the assistance of crystalline insulin is especially useful. The amount of insulin must be adjusted to the patient's needs. Immediately following the operation, adjustment of the diet is necessary. General anesthesia with ether or chloroform should be avoided whenever possible (see p. 157).

Amputation. Before amputation of a part is considered conservative treatment should be attempted. One should aim for (1) proper control of diabetes, (2) control of infection, (3) prevention of trauma and (4) improvement of the circulation. Many sensitive tissues of the lower extremities have been burned by the use of strong antiseptic solutions or the use of too much heat. A warm solution of half saturated boric acid and half 50 per cent alcohol works satisfactorily and its application can be alternated with periods of mild dry heat, as applied in boxes or cradles, wherein the light bulbs are kept at a safe distance from the feet. At the Mayo Clinic the use of the positive negative pressure machines has been unsatisfactory, for there is great danger in that type of treatment if the foot is infected. The care of the skin and circulation of the lower extremities is particularly important in the prophylaxis of complications of diabetes (see p. 450).

TREATMENT OF DIABETES IN THE ELDERLY

The elderly patient who has been suffering from the symptoms of diabetes should be grateful that his condition has been discovered and that adequate and excellent measures for its correction are available.

For the patient whose sugar utilization deficiency was uncovered by accident there is likewise cause for satisfaction, since measures may be followed to control the further course of the disturbance before marked alterations have taken place. The attitude of the patient and his willingness to cooperate with the physician are of first importance. Long established habits of eating, drinking and smoking should certainly not be subjected to any abrupt or radical change. The patient should be given as generous a diet as is compatible with normal nutrition and the metabolic inadequacy covered

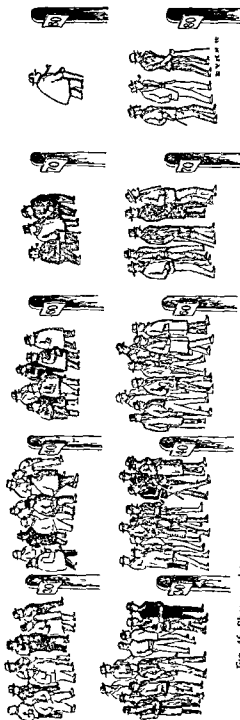


Fig 46 Showing detrimental influence of obesity on longevity (After Joslin from Bortz Diabetes, F A Davis Co)

by insulin Excessively restricted diet prescription can only depress the patient and serve no useful purpose

It is wise to keep in mind that the older one grows the fewer calories one requires (see Fig 46). The metabolic rate declines with age, physical activity requiring calories also diminishes. So far as the optimum amount of carbohydrates intake is concerned, two possibilities exist. First, a moderately liberal quantity can be allowed, second, a high intake of carbohydrates can be given. Recently, more liberal use of carbohydrates has been prescribed by the majority of clinicians, with the hope that the oxidation of the glucose will prevent the development of arteriosclerosis. Likewise, it is easier to regulate the patient's desire for food.

Patients should be impressed with the necessity of accurate measurements of their diet ingredients. For those individuals who require zinc insulin a fourth feeding hour is necessary.

Insulin acts as a most effective tonic. It furnishes the energy that makes life worth living. Far from fearing its use, the patient should be grateful that such an effective remedy is available.

Thiamine chloride, 5 mg three or four times daily, is exceedingly helpful in the treatment of the diabetic.

Bowels should be kept open, if possible by readjustment of diet and exercise. When further remedy is needed, a cathartic is necessary.

Sedation is necessary in some cases. It has been found that glucose varies with the patient's activity. For example, if the level of glucose is high in a diabetic, its utilization is correspondingly increased. When insufficient glucose is oxidized, acidosis supervenes. When the intake is inadequate, other problems arise. So long as the patient is active there is rarely any danger of restricting his glucose intake too sharply. The appetite, under the guidance of energy requirement, usually forces an adjustment. However, when the patient is quiescent or in bed or ill, the danger may be very real.

ARTERIOSCLEROTIC ASPECTS OF DIABETES

According to Keeton,⁵ physiologic senility appears when degenerative changes have limited the individual's physical activities and lessened or abolished his sex life. Pathologically, it begins in extensive arteriosclerotic changes which make it impossible for an adequate distribution of the blood and oxygen to take place in the tissues.

In the sclerotic individual the stage is set for vascular accidents. Great care must be exercised to avoid any sudden changes in the status of the patient. Therefore, sudden and marked restriction in the diet of a patient is usually not good therapy. In any event, great caution must be taken to avoid an insulin reaction due to excessive dosage. A sudden drop in the blood sugar will promptly call forth increased secretion of adrenalin, which is obviously not in the best interests of the patient's cardiac action. In the presence of edema, diuretics necessary to the elimination of the extra fluid should be used. With the elimination of the fluid, however, excessive use of insulin must be guarded against.

Ryegerson³ states that "until the exact relationship between the

sclerosis, it may be better, when any evidence of arteriosclerosis is detected, to avoid diets high in fat. This diet should be sufficiently low in calories to prevent obesity."

If the patient needs insulin he should use enough to keep his urine free from sugar and have his urine tested regularly to make certain that he has accomplished his purpose. Too much insulin should not be given, many physicians are of the opinion that insulin reactions may predispose to the development of coronary occlusion.

Glucose in Diet Woodyatt⁶ pointed out that the glucose in the diet may be increased significantly in elderly individuals without arteriosclerosis without the extra glucose appearing quantitatively in the urine. Elderly patients are contrasted sharply with younger patients, in whom once the glycosuria

there are modifying factors in the tissues themselves which influence the action of insulin, somewhat similar to the loss of insulin activity in patients suffering from acidosis and/or infection.

It is a well known fact that certain individuals appear to be highly sensitive to insulin and others to be insensitive. This variability apparently increases with age.

Hypoglycemia Hypoglycemic states produced by insulin on arteriosclerotic hearts are dangerous.⁵ Experimentally induced insulin shock causes changes in the S T segment with inversion of T waves and conduction deformities in the electrocardiographic tracing. Anginal attacks have been experienced by many diabetics receiving insulin (see p. 406).

Joslin¹ cites a number of cases in which patients have suffered attacks of coronary thrombosis following a rapid fall in the blood sugar. Because of this unfortunate effect, many clinicians were originally skeptical about the use of insulin in elderly patients. At the present time, as emphasized by Keeton,⁵ clinicians are convinced of the value of insulin, but advise extreme care in the avoidance of reaction, however difficult it may be to do. When an arteriosclerotic patient who is receiving insulin shows unusual symptoms a reaction should be suspected.

Within limits, the older the individual the slower may be the reaction from insulin. Therefore, the inexperienced doctor may become impatient and prescribe excessive quantities of insulin thereby inviting a hypoglycemic reaction.

Statistics from the vast material of the Metropolitan Life Insurance Company, compiled by Herbert M. Marks, show that the average age of patients dying from arteriosclerotic disorders has increased (see Table 5).

Arteriosclerosis is looked upon as a degenerative process of the cardiovascular system. It is more commonly found at the present time than in former years because more patients are living into the later years. For this reason it is to be expected that the incidence of coronary disease, cerebral vascular accidents, and peripheral thrombotic processes will increase. The postponement of premature arteriosclerosis is already at hand and one no longer finds arteriosclerosis routinely in all cases of diabetes of five years or more duration.¹

If it is true that the cause of premature occurrence of arteriosclerosis

in diabetes is excessive fat in the body, in the diet, and in the blood, because of insufficient control of the diabetes, then these important deductions are serviceable in planning a regime whereby the onset of arteriosclerosis can be delayed and its effects minimized

During the era in which high fat and low carbohydrate diets were the vogue, arteriosclerosis of advanced degree was frequently observed, particularly in the uncontrolled diabetic patient. With the change in the plan of diet, to include larger quantities of carbohydrate foods, with more limited amounts of fat food, it was surprising to find that less insulin is required to control the diabetic state, and that the arterial system is kept more resilient

TABLE 5

AVERAGE AGE AT DEATH AND AVERAGE DURATION OF DIABETES OF PATIENTS DYING FROM ARTERIOSCLEROTIC CONDITIONS¹ 1930-1932 AND 1933-1935 COMPARED

	Average age at death		Average duration (years)		Number of cases	
	1930-1932	1933-1935	1930-1932	1933-1935	1930-1932	1933-1935
Arteriosclerotic conditions						
Total						
Male	66 1	68 6	13 1	13 5	161	91
Female	65 7	68 5	11 4	13 1	163	124
Coronary artery disease						
Male	64 4	67 6	13 3	16 0	53	31
Female	64 3	68 7	12 0	14 4	41	27
Gangrene						
Male	66 9	68 7	10 8	14 6	25	12
Female	65 7	68 9	14 1	12 1	17	18

¹ Includes deaths from chronic heart disease, cerebral hemorrhage, chronic nephritis, arteriosclerosis, gangrene, etc.

Prepared under the supervision of Mr. Herbert H. Marks of the Statistical Department of the Metropolitan Life Insurance Company, from Joslin, Root, White and Marble, 'Treatment of Diabetes Mellitus.' Lea & Febiger

Some authorities, notably Dragstedt, are of the opinion that arteriosclerosis is a metabolic disturbance of fat metabolism, just as diabetes is a disturbance in the carbohydrate metabolism. They maintain that some hormone necessary to adequate metabolism of the fatty foods is deficient in patients developing arteriosclerosis. If this is the case, then the vast majority of individuals, sooner or later in life, exhibit a deficiency in this particular hormone.

Experimental evidence at hand, from the work of Leary, Page and their colleagues, indicates that complete metabolism of carbohydrate and also of fat in persons who live a temperate existence and who are not overweight is probably as far as one can go today in definite objective efforts to control the development of the arteriosclerotic process.

The most frequent complications due to arteriosclerosis in diabetes are those which occur in the vascular system of the heart and the lower extremities. At the Mayo Clinic arterial insufficiency in the lower extremity

found eleven times more frequently in diabetics than in nondiabetics. It is on this account that exquisite care of the skin of the feet is necessary in order to avoid the possibility of later amputation (see p 453).

Since more and more diabetics are living into later years the complication of coincident heart disease is more common. Coronary occlusion was found by Root, Bland, Gordon, and White⁷ to be five times as frequent in a group of diabetic patients as compared with nondiabetics. In 7000 diabetics Root and Graybill found 210 with angina pectoris. Root¹ is of the opinion that high blood pressure plays a definite role in the occurrence of coronary disease, as it was found to be twice as great among diabetic patients with hypertension as in diabetics with a normal blood pressure. Foster, quoted by Root, found degenerative changes in the myocardium at autopsy in every patient dying of diabetic coma. It is believed that these changes may well be due to dehydration, since the young patients without vascular disease show no clinical evidence of myocardial inadequacy.

TABLE 6*

CORONARY OCCLUSION IN DIABETIC AND NONDIABETIC PATIENTS IN PERCENTAGE OF CASES IN AGE GROUPS¹

Age groups	Males				Females			
	Diabetic		Nondiabetic		Diabetic		Nondiabetic	
	Cases with occlusion	Per cent of total	Cases with occlusion	Per cent of total	Cases with occlusion	Per cent of total	Cases with occlusion	Per cent of total
41-50	2	15	23	7	3	11	2	1
51-60	13	33	47	10	13	25	8	3
61-70	28	48	52	11	26	40	17	7
71-80	9	41	24	11	13	43	9	10

* From Joslin, Root, White and Marble, "Treatment of Diabetes," Lea & Febiger.

¹ Root, Bland, Gordon, and White. JAMA 113:27, 1939.

Root and Sharkey found acute thrombosis, with infarction of the heart, in 21 per cent of 175 autopsies of diabetics. Rupture of the heart occurred twice and there was an aneurysmal dilatation of the wall of the left ventricle twice in this series.

the blood sugar often rises and the amount of insulin required is increased. Oftentimes the diabetes may become very mild in those patients in whom cardiac or nephritic decompensation occurs. With the elimination of the edema, through the use of strong diuretics, the glycosuria may return.

Vascular accidents in the form of embolic or thrombotic occlusions

have, during the past few years, been treated by the use of anticoagulants with gratifying success. The early use of heparin and dicumarol, plus papaverine, has been effective in controlling occlusions, within the heart itself and also in the extremities. The routine for dicumarol therapy has been outlined by Falk¹¹ as follows:

"1 Patients must be hospitalized and dicumarol therapy must not be attempted unless it is certain that the laboratory is prepared to make accurate prothrombin readings, which must be done each morning before the day's dosage of the drug is given.

"2 Preliminary management consists in the administration of papaverine intravenously, with oxygen and morphine if necessary.

"3 After the diagnosis is established, check the prothrombin level and give 300 mg of dicumarol by mouth.

"4 Repeat the dosage of 300 mg daily after the morning prothrombin level is determined, until 50 per cent is reached.

"5 After 50 per cent is reached, give 100 mg of the drug each morning until 35 per cent is attained.

"6 Now stop the medication for several days (as the level is likely to drop lower without further dosage). Thirty five per cent is the stop signal."

"7 When the level again rises above 35 per cent, give 50 to 100 mg of the drug daily, attempting to hold the level between 35 per cent and 50 per cent.

"8 Keep this level for four to six weeks, as long as the patient is in hospital.

"9 If the level gets down to 15 per cent (rare), watch for hemorrhagic phenomena, such as red cells in urine, petechial spots or purpuric areas. Treatment: Give 60 to 72 mg of synthetic vitamin K intravenously, give the second dose in four hours.

"10 In the event of alarming hemorrhage, give FRESHLY citrated blood as often as needed. Blood loses its prothrombin in from twenty four to thirty six hours."

Allen¹² has described the experiences at the Mayo Clinic. He believes that, if treatment is instituted early and followed through courageously, many patients will be saved the distressing experiences of venous insufficiency. The early and judicious use of penicillin and the sulfonamides in the conservative treatment of infections of the feet, even in the presence of a seriously compromised circulation, has resulted in a striking reduction in the necessity for amputation in our experience. Also, infections of the toes resulting from impaired circulation in the smaller vessels have responded to anticoagulant therapy and the need for surgery has been reduced. Surgical procedures where indicated have been of a much less radical nature.

Until the advent of insulin and its practical use in the treatment of diabetes, patients ordinarily did not live sufficiently long to develop advanced changes in the cardiovascular system. At the present time, the diabetic lives and dies in the arteriosclerotic zone. The average age at death of 927 cases studied by Root¹ was 64.8 years. As a cause of death in the diabetic patient, arteriosclerosis has risen threefold in importance, while coma has dropped to one sixteenth of its former importance.

Notes have appeared in the literature warning against the use of insulin in the diabetic who has manifestations of vascular degenerative disease.

because of the danger of precipitating vascular spasm or thrombosis during hypoglycemic attacks. Vascular crises are recognized to occur most commonly in arteries that have undergone sclerotic degeneration as a result of essential hypertension or progressive involution with aging (see p. 468). Reports in the literature indicate that these sclerotic lesions are a more frequent phenomenon in the diabetic than in the nondiabetic, and in the poorly treated diabetic than in the well controlled diabetic.⁸

TABLE 7

INFLUENCE OF DURATION OF DIABETES UPON PERCENTAGE OF TOTAL DEATHS IN DIABETICS DUE TO ARTERIOSCLEROSIS AND DIABETIC COMA¹

Epoch	Average duration of diabetes, yrs	Deaths, total	Coma, per cent	Arterio-sclerosis, per cent	Average age at death, yrs
Naunyn 1894 to June, 1914	4.9 ²	325	64	18	44.8
Allen June, 1914, to Aug., 1922	6.1 ²	835	42	25	45.3
Banting Aug., 1922, to Dec. 31, 1925	7.6	535	22	42	53.9
Jan., 1926, to Dec. 31, 1929	8.4	899	11	50	60.0
Jan., 1930, to Mar. 13, 1935	11.0	981	6	56	62.8
Hagedorn Jan., 1937, to Mar. 29, 1940	12.5	927	4	60	64.8

¹ Prepared by the Statistical Bureau of the Metropolitan Life Insurance Company, from Joslin, Root, White and Marble, "Treatment of Diabetes Mellitus," Lea & Febiger

² Based on cases with known duration

Case example. White male, age forty-seven, was admitted to the hospital complaining of attacks of precordial pain radiating to the left arm. Physical examination disclosed no palpable abnormality. Electrocardiogram showed left axis deviation. He was given a diet of C 150, P 75, F 70. His glucose tolerance suddenly broke and this was followed by an aggravation of his stenocardial syndrome. A reduction in diet to C 90, P 60, F 70 without insulin controlled his attacks.

Insulin. In the use of insulin as a therapeutic agent the following principles are employed to protect the patient against even the mildest form of hypoglycemic reaction:

1. *Dietetic Control.* The caloric value of the diet should be equivalent to the total energy requirements of the body. Protein is to be given in the proportion of 1 gm. per kilogram of body weight. Carbohydrates are best prescribed in amounts equal to the lower limits of what the normal person

takes Fat intake is established by subtracting the caloric value of the protein and carbohydrates from the total and dividing the figure by nine

2 *Insulin* Insulin is started in small doses which are gradually increased up to the point where the sugar disappears from the urine entirely or is present only in a slight trace

3 *Dietetic Measures* To offset insulin shock, 15 gm of readily absorbable carbohydrate (preferably in the form of fruit juice) is given the patients two hours after the dose of insulin This procedure is most important

The result of such management has been that hypoglycemic attacks have become a rarity No cases have been made worse by the treatment

According to Hyman and Parsonnet⁹ anginal symptoms are frequently aggravated by the use of insulin They suspect that insulin has a toxic effect upon the myocardium regardless of its relationship to the metabolism of glucose In the final analysis, insulin, like any other pharmacologic substance, may be a toxic or a therapeutic agent depending upon the manner of its use

PROPHYLACTIC GERIATRICS

Diabetes mellitus is a metabolic disorder characterized by an increase in the blood sugar, which may or may not result in sugar appearing in the urine With a high renal threshold, such as many individuals in the later years of life may exhibit, glucose may be present in abnormal quantities in the circulating blood stream and not "spill over" into the urine

Conditions such as profound sepsis, fevers, accidents, and increase in the metabolic rate may activate a latent diabetic state into an active one It is likely that diabetes may remain latent, and in fact never become active, in the absence of sufficient strain of the metabolic function

Overweight and overeating subject all of the organs and tissues to an undesirable strain The greater the overweight, the greater is the strain, and particularly is this true of the digestive and metabolic function and the insults thereby incurred upon the pancreas

The noted columnist, Arthur Brisbane, once wrote, 'half of what we eat keeps us alive, the other half keeps the doctor alive' Some authorities are of the opinion that nearly 50 per cent of the ordinary ailments that send folks to their doctors arise from dietary indiscretions and notably from over-indulgence In addition to subjecting body organs to inordinate and unnecessary strain, overweight is a definite hazard to individuals in the later years of life¹⁰ Particularly from the age of fifty years onward obesity may be regarded as a threat against sound and vigorous health

There is an optimum weight for each individual above which unnecessary increase predisposes to various disease processes and below which the individual will not possess the vitality and stamina that should be his While the insurance tables regarding the ideal weight per person per sex per age are only approximate they prove helpful in suggesting within five or ten pounds just what a person should weigh (See p 35)

Naunyn and Von Noorden were the first to point out that improvement in the diabetic on the restricted diet was due to rest of the pancreas Some observers do not agree with this conclusion Allen showed that starvation

usually mild when uncomplicated. The diabetic state is easily controlled during an initial period of intentional undernutrition. When weight is lost there follows a remarkable recovery of carbohydrate tolerance, which eventually allows an increase in the diet to maintenance levels containing 200 to 225 gm of carbohydrates. This same type of patient, if given insulin at the onset of treatment, with a diet that is not too large, will also regain tolerance, so that often the insulin can be discontinued eventually without a return to the diabetic state when the patient remains on diet alone. If this type of adult obese diabetic patient does not restrict his food consumption the hyperglycemia and glycosuria persist despite large amounts of insulin.

Obese persons who have had their diabetes for a long time and have remained overweight either because of previously prescribed high caloric diets or because of neglect in treatment, seem to lose their ability to regain carbohydrate tolerance. They usually continue to require insulin to "cover" the food consumed.

Those patients whose diabetes manifests itself after the age of forty and who are underweight or of normal weight when they come to the physician are either (1) cases in a later stage of the obese type, (2) adults forty to sixty years old who were never overweight, or (3) senile asthenics over the age of sixty. The first group requires small doses of insulin at the onset of treatment, in time their tolerance for carbohydrates will improve, thus allowing later dietary increases without change of insulin dosage. The senile asthenic patient with diabetes has a mild case and usually does not require insulin. Here higher fat diets can be used to good advantage. The best treatment of the other non obese adult persons with diabetes does not seem to follow a definite pattern.

As diabetes is generally hereditary in nature, children of diabetics should be particularly careful in observing the ordinarily accepted rules of diet and personal hygiene.

While there is general agreement on the phrase "Once a diabetic, always a diabetic," it is the common experience of physicians treating large numbers of diabetic patients that after the patient has strictly followed the dietary prescription either with or without insulin and exercised according to his needs, the blood sugar will maintain itself within the customary limits of normal and the patient will experience none of the symptoms or disturbances that the uncontrolled diabetic is annoyed with.

PROGNOSIS

The efficiency of the pancreas so far as manufacture of insulin goes, probably varies. Under long continued strain from overeating or other causes the pancreas becomes fatigued or exhausted and the sugar stored in the liver, and the customary sequelae appear.

The first principle in the treatment of a diabetic at any age is metabolic rest. The pancreas must be given an opportunity to recover its functional efficiency in the manufacture of insulin. A markedly restricted diet, planned to meet only the minimum requirements of the body, is prescribed so that the least possible demands on the pancreas are made. Frequently insulin administration is necessary for a variable period of time. However, after

some days or weeks of treatment under ideal conditions the pancreas often has so recovered its ability to manufacture insulin that administration of insulin may be discontinued. This fact suggests a remission of the pathologic physiology. However, whether this process ever reverts to a point which might be regarded as "cure" of the diabetic condition has thus far not been admitted by any investigators.

The brilliant researches of Young and Lukens, whereby diabetes has been produced in the experimental animal by the injection of an extract of the pituitary gland, have permitted study of the pathologic changes occurring in the pancreatic gland. Until these pathologic lesions have reached the end-stage of fibrosis it is possible to reverse the changes by the use of insulin and a diet rich in fats. These investigators are of the opinion that the process is reversible and the animal can be cured of his experimental diabetes. These investigations mark another milestone in man's battle against disease. Its significance for the human patient is yet to be proved, but it is likely to be very great.

Case example. A minister, age fifty-five, white, and in supposedly good health, was injured in a railroad accident. Soon after that he commenced to lose weight, worried a great deal, became very nervous, lost twenty-five pounds in a few months. Examination of the blood sugar showed 265 mg. per 100 cc. Patient was placed at bed rest with a diet of C 150, P 70 and F 120 in four feedings. He received protamine zinc insulin. In a period of two weeks time the blood sugar was brought to normal and the patient started to gain weight, and the symptoms disappeared entirely. However, he continued to use 10 units of insulin twice daily for several months. Gradually the insulin was reduced until the patient

Many patients have a diabetic condition of so mild a degree that they require insulin for a relatively short period of time. The pancreas apparently regains sufficient of its function to furnish the body with enough insulin so long as no excessive demands are placed upon it.

Frequently there is a mild depression of the metabolic activity in individuals in the later years of life. While not definitely diabetic, elderly individuals oftentimes show retardation in their utilization of carbohydrates. These persons are benefited by wholesome regulation of the diet, plus small quantities of insulin. There is often a marked increase in their well-being, the patients taking on a new zest and joy in living. For them insulin acts as a shock absorber, as a tonic. It protects the pancreas, which is evidently tired, and gives the gland a chance to recover its former strength and ability to manufacture insulin.

SUMMARY

The geriatrician needs to regard the diagnosis and adequate treatment of his diabetic patient as a growing problem. With this increasing responsibility, however, augmented opportunities exist for improving the vitality and vigor and also probably the life span of this important portion of the population.

In nearly every instance when it is suspected, diabetes may be readily diagnosed. The care of the elderly diabetic includes wholesome measures of

personal hygiene with due attention to nutrition, elimination, and rest. The study of diabetes in its broader aspects and details equips the practicing physician with information of inestimable value in treating all patients that come within the scope of the geriatrician's experience

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CHAPTER 15

GOUT

JOHN H. TALBOTT

INTRODUCTION

ALTHOUGH the first attack of acute gout may appear in the earlier decades of life, chronic deforming changes from gouty arthritis are seen frequently in the advancing years. It seems appropriate, therefore, to discuss this malady in a comprehensive symposium on geriatrics. No attempt will be made to reconcile the several conflicting presumptions in the literature regarding this malady,¹ nor will a general survey of the literature be included. A comprehensive review has recently been presented² and the reader is referred to this for more detailed information. The conclusions presented at this time are those of the author in most instances and are based upon observations collected in his laboratory and clinic. A survey of fifty patients with gouty arthritis, each fifty years or older, constitutes the basis of the clinical material. The inferences from this group are similar to those obtained from a larger group of patients of all ages, investigated at the Massachusetts General Hospital from 1934 to 1942 and at the Buffalo General Hospital since 1946.

DEFINITION

Several adjectives have been employed to qualify the term gout or have been used to describe the clinical syndrome and its variations in current medical literature. It is believed that clarification will be achieved if the nomenclature in this disease is defined strictly. Gout is the parent term and a patient is afflicted with the malady irrespective of the stage of development of symptoms. Acute gouty arthritis or an acute attack of gout refers to the periodic attacks of acute joint distress in patients with gout. Intercritical gout is the period between attacks of acute arthritis irrespective of the presence or absence of deforming changes. Chronic deforming arthritis is a late manifestation. Several decades may elapse after the first attack of acute gout before this stage is reached.

INCIDENCE

Gout is not a rare malady and it is agreed by several observers that patients with gouty arthritis constitute at least 5 per cent of all patients suffering from nonsurgical joint disease. The belief in its apparent rarity arises from the failure to recognize the disease prior to the development of

society and there is meager support for the presumption that gout is a disease of persons who are habitually intemperate whether this refers to alcohol

food, tobacco, or sex. Most of the patients are males and the sex ratio of afflicted persons is approximately 20 to 1 in their favor. Particular caution should be exercised in confirming the suspected diagnosis in a female, so infrequently are they affected. Gout is widely distributed throughout the world and shows no particular preference for nationality or race. The familial incidence is high if careful attention is given to this aspect. A positive family history is frequently of help in confirming the diagnosis of a suspected case.

CLINICAL DESCRIPTION

The Acute Attack. Gout rarely produces symptoms prior to the onset of acute articular distress. An acute attack of gout is usually the first sign of this metabolic dyscrasia and the diagnosis is not suspected until after this event. The metatarsal phalangeal joint of the great toe frequently is the first joint to be affected. Symptoms of an acute attack of gouty arthritis appear suddenly at any time of the day or night and may reach a maximum of intensity within a few hours. Rarely is the onset protracted and of slow development. Acutely affected joints show the cardinal signs of inflammation—redness, swelling, tenderness, and heat. A septic joint may be suspected on examination. Inflammation with involvement of the lymphatics may extend beyond the immediate vicinity of the affected joint. It is wise to be cautious in recommending surgical treatment of an acutely inflamed joint of unknown etiology in view of the possibility of acute gout. The pain in the joint may be excruciating and well-nigh intolerable. Fever and leukocytosis accompany the involvement and are evidence of a constitutional reaction to an acute process. Pain is caused in part by an effusion into the joint space and edema of the surrounding soft tissues. Tenderness is usually maximum on the lateral aspects of involved joints. The attack may be mono-articular or migratory polyarticular. Although the involved joint appears red in color, it may have a purplish hue which is of aid in distinguishing it from a septic joint.

Acute attacks of gout tend to involve only articular structures, although nonarticular tissues may be affected independently of a neighboring joint. Subcutaneous bursae infiltrated with sodium urate or subcutaneous tophi may be acutely involved and extremely painful. Large joints, notably the knee, may be the site of a large effusion and may require one or more aspirations for the relief of pain. Roentgenograms of acute gouty joints are similar to those taken between attacks except for the soft tissue swelling.

The duration of an attack depends upon several factors. Symptoms may continue for many days or even for several weeks if specific measures are not instituted. Occasionally a patient is seen with severe joint symptoms which have persisted for several weeks. This may be caused by a series of single attacks or by one prolonged episode. On the other hand, if colchicine therapy is begun at an early hour, the acute articular symptoms respond promptly and, if there is no deformity, there are no residua except possibly soft tissue swelling which may persist for some time. Desquamation may be the final mark of a recent attack. If specific measures are not instituted until several days after the onset of acute symptoms or at times until after several weeks, the response of acute joint symptoms to colchicine therapy is not so prompt and additional medicine may be necessary.

The interval between the first and second attack may be as long as a

decade or as short as a few weeks. It usually exceeds twelve months. The interval tends to be shorter as the disease progresses and eventually there will be one or more attacks each year. Chronic deforming changes may develop by this time.

Chronic Deforming Gout. The boundary line between acute and chronic gout is not always definitive although the diagnosis of the latter is usually justified when diminution of function of affected joints persists in the inter-critical period. Chronic deforming gout does not imply suffering and may be manifest by little more than limitation of motion of one or more joints. The development of deforming changes which is observed in persons in the latter decades of life is usually the result of many acute attacks over a period of years. We have never observed a patient with demonstrable permanent damage following only one acute attack.

The majority of the joints of the hands and of the feet, arms, and legs may be involved in some patients. Occasionally the shoulder, sacroiliac or sternoclavicular joints are invaded. There may be complete ankylosis of several joints in advanced cases. Eventual commitment to bed as a permanent cripple because of chronic gouty changes is the fate of less than 5 per cent of all afflicted persons. Seventy four per cent of the patients in this series of fifty showed changes of the joints of the extremities which were interpreted as osseous tophi. The joints of the feet, usually the metatarsal phalangeal joints of the great toe, have been the site of one or more attacks in each patient. All except nine have had acute attacks of gouty arthritis in the knee, elbows, wrists, or hands as well as in the feet. Five of the patients are over eighty years of age and not one of these is bedridden with gouty arthritis.

PRECIPITATING FACTORS

Gout is an hereditary metabolic malady and is believed to be caused by other factors than food and drink. Indiscretion in eating and drinking may precipitate an acute attack in an occasional patient but is believed to have little to do with pathogenesis of the disease. There are several agents which have a much closer relationship to the acute attack. Certain drugs may precipitate an acute episode. Liver extract with its high purine content may be an offender. Salyrgan, insulin, and ergotamine may do it occasionally. Direct trauma frequently is responsible. Poorly fitting shoes or tight gloves may do sufficient damage to produce acute symptoms. Acute infections are dangerous associates of gout. An acute pharyngitis, sinusitis, or other type of

any patient with gout following a surgical procedure whether it be minor or major.³

DIAGNOSIS

of symptoms between the attacks, (2) subcutaneous urate tophi, (3) osseous tophi, (4) increased concentration of serum urate, (5) response of acute symptoms to colchicine, (6) family history of gout, and (7) the excretion of urate calculi.

History A history of intermittent attacks of acute articular distress with absence of joint symptoms between the attacks is highly suggestive of gouty arthritis. If the gout is complicated by nonspecific chronic degenerative



Fig 47 A Tophus on ear B Large subcutaneous tophus on elbow

changes as it may be in older persons, there may be some interval discomfort but this should not be attributed primarily to gouty changes. With advanced



Fig 48 X ray of elbow shown in Fig 47 B. No invasion of periosteum by tophus

articular changes, interval symptoms are to be expected irrespective of the age of the patient or the duration of the disease.

Urate Tophus A subcutaneous urate tophus (Fig 47) is the hallmark of chronic gout but it is not the *sine qua non* of gouty arthritis. Tophi do not

develop usually before the first bout of gouty arthritis. Tophi have a predilection for articular structures such as synovia, cartilage, tendons, and certain nonarticular cartilage particularly the helix of the ear. Patients who have widely disseminated tophi usually have extensive involvement of bone as well.

The appearance and recognition of a subcutaneous tophus are conditioned by its age, location, and depth under the skin. The color may be pearly white or it may appear yellowish. If the tophus is in an exposed location and traumatized easily, a chronic sinus develops with seepage of urate. In protected regions, tophi may attain considerable size with little or no



Fig. 49 Sodium urate needles from aural tophus

change in the overlying skin. The larger and older the tophus, the more desiccated is the contained urate salt. Large tophi may be as firm as calcium deposits and may be mistaken for calcinosis. Identification of the contents of a suspected urate tophus is not difficult and the procedure is to be strongly recommended. A small amount of chalky material is aspirated by a hypodermic needle and syringe and is placed on a glass slide. Examination under a microscope reveals needle crystals which are characteristic of sodium urate (Fig. 49).

Ossseous Tophi. These appear as punched out areas by x-ray. Calcium salt is replaced by sodium urate and increased radiability of the involved area

follows. Tophi must attain a considerable size, perhaps 5 mm in diameter, before they are visible by x-ray. The x-ray examination usually is negative in the first years after the onset of an acute attack, save for soft tissue swelling during the acute episode. The presence of demonstrable tophi in bone is indicative of irreparable joint involvement and is evidence that the joint has been the site of several acute episodes. The usual sites for osseous tophi are the heads of the metatarsals and the areas immediately adjacent to the phalangeal joints of the fingers and toes. Bones which are affected less frequently include clavicle, scapula, ulna, radius, humerus, carpals, ilium, sacrum, vertebrae, patella, femur, fibula, tibia, and tarsals. In gouty patients who are more than fifty years old, the structural changes observed in degenerative joint disease are especially frequent.

In spite of the several changes noticed at x-ray examination, a diagnosis based upon these findings alone is hazardous in any patient suspected of having gout. If the bony structures are normal by x-ray, this may mean little more than that extensive deposition of sodium urate has not occurred. Many

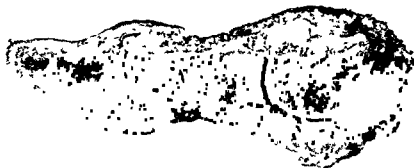


Fig. 50. Sagittal section of amputated great toe.

years may elapse in mild cases before secondary changes are visible at x-ray examination. On the other hand, it is appreciated that other chronic conditions such as rheumatoid arthritis and degenerative joint disease may have punched out areas in the bones of the hands or the feet which may make roentgenographic interpretation difficult. Less common maladies which show certain osseous changes by x-ray which have a similarity to those of gouty arthritis include cystic disease of bone, multiple myeloma, hyperparathyroidism, Boeck's sarcoid, carcinoma with bone metastases, syphilis of the bone, leprosy, yaws, tuberculosis, and late effects of chronic trauma of the feet.

Serum Urate.* The concentration of serum urate is elevated in gouty persons irrespective of the presence of acute or chronic symptoms. This diagnostic procedure is particularly significant if the patient is seen at the

time of the first or one of the earlier attacks and prior to the development of subcutaneous or osseous tophi. Concentration of urate in blood is not an infallible diagnostic guide⁴ but it probably is as reliable as the determination of a basal metabolic rate in a suspected case of thyrotoxicosis.

The determination should be done on serum or plasma rather than on whole blood.² Serum or plasma is to be preferred to whole blood in the determination of most constituents. Many of the chemical constituents of the blood are distributed unequally between serum and cells, any fluctuations in cell volume will affect the final result on whole blood even though no change in concentration of the constituents in either cell or serum phase has occurred. Avoidance of cellular substances, which interfere with the color development in the urate procedure, is an additional argument in favor of the use of serum. The range of uric acid concentration in serum or plasma is somewhat above that reported for whole blood. The upper range for whole blood is approximately 4 mg per 100 cc and for serum or plasma approximately 1 mg higher. Experience has shown that most nongouty persons have a serum urate level of less than 5 mg and most gouty persons have a level greater than 6 mg. There is a supplementary advantage, therefore, in using serum due to the intervening range of 1 mg between 5 and 6 mg which separates many nongouty individuals from gouty patients.

Conditions other than gout which may be associated with an elevated uric acid content of the serum include leukemia, polycythemia vera, pernicious anemia, lead poisoning, starvation, renal insufficiency, and certain acute infections such as pneumonia. The elevation of serum urate in most of these maladies is of academic interest in so far as the differential diagnosis of gout is concerned except for renal insufficiency. Since renal involvement is a common accompaniment of gout, it is important that caution be exercised in interpreting the urate level in any patient with joint disease and renal insufficiency. This is particularly applicable to patients past the age of fifty who may develop incipient renal insufficiency as an integral part of the life cycle.

The urate level may be influenced by certain drugs and the forcing of fluids. If the concentration in a gouty subject is at the lower range, a large fluid intake may depress temporarily the level below 6 mg. Diuretics such as salyrgan may flush out a significant amount of urate and decrease the concentration 2 or more mg per 100 cc. Cinchophen and salicylate have a similar effect and their action may persist for two or three days. In interpreting the level of serum urate, attention should be given to these possibilities.

Response to Colchicine. Full doses of colchicine in the treatment of an acute gouty joint are of diagnostic significance as well as of therapeutic value. Inadequate amounts of colchicine provide little symptomatic relief and may give, therefore, a misleading diagnostic answer. Other forms of acute arthritis are affected little or not at all by colchicine.

Family History. A positive family history for gouty arthritis possesses confirmatory value but is not diagnostic. Nevertheless, if there is a positive family history in a patient with unexplained acute arthritis, the possibility should be considered. Furthermore, the study of an unexplained acute joint is not complete without careful attention being given to the familial medical background.

Urate Calculus. The passage of a urate calculus has been observed in a

few instances preceding the first attack of acute arthritis. Inquiry should be made into whether or not the patient has had renal colic or a renal stone and whether or not it was analyzed for its chemical content.

DIFFERENTIAL DIAGNOSIS

The several arthritides offer the chief difficulty in the clinical differentiation of gouty and nongouty disturbances.⁵ Acute rheumatic fever may be confused with gouty arthritis in younger persons but the incidence of the former in the declining decades is low. Rheumatoid arthritis has a higher incidence among females than males, gout has the reverse sex ratio. Constitutional symptoms such as malaise, weight loss, and vasomotor disturbances usually are seen in rheumatoid arthritis. The arthritis is more symmetrical, the duration of single attacks much longer, and the onset more insidious than in gout. Patients with rheumatoid arthritis do not respond to colchicine. Concentration of uric acid in the serum should not be elevated in patients with rheumatoid arthritis except when there is a complicating renal condition. Gonorrheal arthritis may be accompanied by articular attacks that are as acute as are encountered in gout. The chronicity of untreated gonorrheal arthritis is a distinguishing feature. The history of a genitourinary infection should be of help. Gouty arthritis does not respond to the administration of sulfonamides or penicillin as does gonorrheal arthritis. Acute cellulitis of the hands or feet may be confused with gouty arthritis. The content of uric acid in the serum should be determined if possible before any surgery is considered in such cases. Constitutional symptoms may be observed in either condition. Acute cellulitis should respond to antibiotics, these are ineffective in gout. Degenerative joint disease, which is the commonest form of arthritis in the older age groups, is usually not associated with acute articular episodes. Females are more often afflicted with degenerative joint disease than males. An acute Heberden's node has a certain similarity to a small gouty tophus and a chronic node may be mistaken on casual observation. The identification of sodium urate from a suspected node is extremely helpful.

PATHOGENESIS

Gout is classified as a metabolic dyscrasia according to modern nosography. This description is somewhat misleading since the etiology is unknown. However, until further work is done which proves or disproves this assumption, the implication is not considered to be particularly harmful. In our attempt to interpret the pathogenesis of metabolic gout, which is believed to be the precursor of gouty arthritis, we have assumed that the increased concentration of urate in body fluids is the basic disturbance. Precisely how the concentration becomes increased is not known.

Role of the kidney. The inability of the kidneys to eliminate urate is a reasonable explanation of the increased concentration of this substance in

inability of gouty patients to excrete urate in the earlier years of the disease must be a functional rather than an anatomic disorder of the kidneys. Since

urate appears in glomerular filtrate in essentially the same concentration in which it exists in plasma, an increased reabsorption of urate by the tubules would be the only mechanism whereby body retention would result. This mechanism is possible, but it seems to us to be unlikely. Secondly, the differentially increased reabsorption must be the only dysfunction operating, for gross inability to excrete other substances normally handled has not been found in gouty patients during the years immediately after the diagnosis was first made. Furthermore, if the kidneys were responsible for the increased

TABLE 8

QUANTITATIVE MEASUREMENTS OF RENAL FUNCTION IN FOURTEEN GOUT PATIENTS

Patient	Serum non-protein nitrogen	Maximum specific gravity of urine	Excretion of phenol sulfon-phthalein during first 15 minutes	Inulin	Creatinine	Intravenous pyelography, interpretation
	mg per 100 cc		per cent	cc of plasma cleared per minute		
Average range for normals	20-35	1.020-1.035	25-35	100-140	135-175	
M S	28	1.016	18	74	88	Left kidney not visualized
T R	42	1.012	5	31	42	
T Y	50	1.014	12	56	80	Incomplete filling of calices
S N	32	1.022	30	95	142	Normal
N O	26	1.010	14	62	84	Slow excretion of dye
D E	32	1.018	18	73	96	Calices indistinct
Y O	44	1.014	5	25	36	Slow excretion of dye
P O	28	1.012	28	72	93	Small right kidney, stones in calices bilaterally
H R	30	1.010	26	69	87	Pelves poorly outlined
E Y	70	1.010	4	12	34	Small kidneys; dye excreted slowly
R R	34	1.020	26	95	137	Normal
S L	28	1.018	18	71	99	Incomplete filling of calices
N S	28	1.016	16	67	75	Normal
Y N	30	1.014	26	102	128	Normal

concentration of serum urate, they would be implicated doubly in patients who developed signs and symptoms of renal failure terminally. Renal failure in such patients is presumed to be largely mechanical following deposition of urate in the tubular lumina. Finally, the most cogent reason for our refusing to accept the renal dysfunction theory of the pathogenesis of gout is the failure to obtain experimental proof in support of it.

In a study of gouty patients, urate clearance was measured simultaneously with clearance of inulin or mannitol and clearance of diodrast or para-

aminohippurate. These are considered to be the most precise techniques available for the measurement of various functions of the normal and diseased kidney. There were several patients in a large series of gouty and nongouty persons who demonstrated a glomerular filtration rate and a renal blood flow within the average range for normals. Urate clearance also was within the normal range. Other patients who had had symptoms of gouty arthritis for a decade or more had varying degrees of depression of glomerular filtration rate and renal blood flow. There was, however, no differential inability of a serious degree in the clearance of urate in any of the patients who showed no other evidence of renal insufficiency by the routine clinical methods, such as ability to concentrate solids or ability to excrete phenolsulfonphthalein after intravenous injection. In a few patients who had renal impairment that was clinically apparent, there was some impaired clearance of urate. Only in such patients as show serious depression in glomerular filtration rate is retention of urate from a kidney disturbance manifest.

Destruction of Urate by the Body A second explanation for the increased concentration of urate in body fluids of gouty patients is that destruction by uricolytic enzymes is impaired. This theory is weak since the body normally disposes of only insignificant quantities of urate by enzymatic activity. Furthermore, it has been shown that uric acid injected intravenously into gouty patients is removed as rapidly as it is in normal persons. Repudiation of the diminished destruction theory, therefore, seems legitimate.

Increased Urate Formation If the first two theories of increased urate concentration are rejected as unsatisfactory, there remains a third, *i.e.*, increased formation of urate by the body. This explanation seems to us to be the most attractive and is supported by experimental evidence from our laboratory. The confirmatory data were obtained from the study of urinary excretion of urate by gouty patients, normal controls, and from a study of the concentration of serum urate of nonaffected relatives of gouty patients. Normal renal function was demonstrated by routine clinical tests in each subject so that none of the observed findings may be attributed to gross renal disease or renal insufficiency. The gouty patients and normal controls were maintained on a low purine diet in the metabolism ward. The average daily excretion of urate by the gouty patients was at all times equal to that of the normal controls and on several days surpassed it. It was concluded that in gouty patients without renal damage, the ability to form and to excrete urate may be increased above normal. The observations on nonaffected members of gouty families revealed an increased concentration of urate in the serum in approximately 20 per cent of persons who gave a negative history for acute arthritis and showed no evidence of joint changes by x ray. The elevated serum urate as observed in symptom free members of gouty families was interpreted as an essential part of the gouty diathesis and is believed to be produced by an increased formation of urate by the body.

PATHOLOGY

The distinguishing pathologic feature of gout is the deposition of sodium urate in soft tissues and in bony structures (Fig. 50) in various regions of the body.⁶ It is believed by the writer, although it has not been proved, that the deposition of urate in joints probably precedes clinical symptoms of acute gouty arthritis. Recurrent deposition of microscopic amounts of urate may

continue for years before tophi are demonstrable grossly or on physical examination. The mechanism responsible for the deposition of sodium urate has not been defined. Undoubtedly it is related to the increased concentration of urate in synovial and other body fluids. Precipitation of salts from body fluids is always a potential threat since the maximum solubility of urate is not great. The fact that the earliest changes observed in microscopic examination of joints are deposits of urate in the upper layers of cartilage suggests that urate comes from synovial fluid rather than directly from capillaries in the bony structure. Deposition of urate and infiltration in the cartilage may occur without demonstrable evidence of necrosis either in the midst of the urate mass or along the outer fringes of the advancing urate border. Urate deposits are prone to develop in avascular rather than vascular tissues. The common sites are articular cartilage, epiphyseal portions of bone, synovial membranes, bursae, ligaments, tendons and the cartilage supporting the helix of the ear, respectively. All of the articular and periarticular structures of several joints may be riddled by urate tophi in advanced cases of gout. Tophi are conspicuously absent from muscles, liver, spleen, lungs, and nervous tissue.

A joint in a young or middle aged person, which has been the site of only a few attacks of acute gout, may show little except microscopic urate deposits. These spread as the disease progresses, and eventually the articular cartilage may be covered as completely as an icing covers a cake. There may be lipping and grooving in older persons, evidence of degenerative joint disease. In an advanced case of gout, urate deposits may be so intermingled with pathologic changes characteristic of degenerative joint disease that differentiation is difficult or impossible. There may be pathologic changes in other areas similar to those observed in rheumatoid arthritis. This has led some investigators to designate gouty arthritis as a mixed type of arthritis. The synovial membrane may show few effects of urate invasion or there may be considerable hypertrophy and loss of normal architecture. Extensive erosion or destruction of the cartilage may be followed in some instances by fibrous ankylosis. Complete destruction of the joint and replacement by a urate tophus rather than by fibrous tissue is even rarer. The few joints that have been observed at x ray examination that have undergone this series of changes have been surrounded by a thin shell of calcium. In other joints, calcium deposition is not the characteristic finding. Absorption of calcium rather than deposition is the rule.

Pathologic studies give little clue as to the mechanism of an acute attack of joint distress. A bulging urate deposit in an outer layer of cartilage rupturing into a joint space before an acute attack has been presumed. Clinical symptoms of acute gout would be caused then by the reaction of the joint to this insult. This is an ingenious explanation and may be valid for acute attacks which follow joint trauma. It does not explain, however, acute attacks which presumably are incited by nontraumatic causes such as infection, a surgical operation, or intemperance.

The pathologic features of the kidney in gouty persons are constant in regard to the presence of urate but variable in the nature and amount of parenchymatous changes. The kidneys may be normal in size and weight, or they may be small and atrophic and less than one third of the normal size. The renal capsules may strip easily or with some difficulty. The exposed

surfaces show minimal to marked scarring. If urate deposition is slight, it will be evident only from the gritty sensation with the knife as the kidney is sectioned. Urate deposits are visible readily on the cut surfaces of the kidney in advanced cases. They appear as streaks of white throughout the parenchyma but are most abundant in the medullary region.

The lesions are as variegated microscopically as they are grossly, and they may simulate chronic glomerular nephritis, vascular nephritis, pyelo-nephritis, or amyloid nephrosis. Glomeruli show complete or partial hyalinization. Many glomeruli which apparently have not lost their ability to function, show thickening of the intercapillary substance. Large and small vessels show proliferation of the intima and narrowing of the lumina. The tubules are dilated and loaded with urate crystals and hyaline casts or are atrophied and surrounded by interstitial fibrosis. Deposits of urate in the interstitium may invoke a chronic inflammatory reaction with accumulation of lymphocytes and foreign body cells. There is no satisfactory explanation for the lack of uniformity of the morbid processes. The presence of vascular disease of the kidney is not unexpected since patients with gout tend to develop arteriosclerosis throughout the body prematurely. The incidence of other types of nephritis may be related to a lower susceptibility of the kidney, which in turn is related to the damage from urate deposition in the renal tubules and parenchyma.

COMPLICATIONS AND MORTALITY

These subjects may be discussed conveniently together. Renal disease is the principal complication of gout, if it is assumed that joint involvement is an inevitable accompaniment of the malady rather than a complication. Furthermore, renal failure is one of the principal causes of death in gouty patients who die prematurely. In the older age groups, coronary and cerebral vascular disease share honors with renal insufficiency.

The usual clinical tests of renal function in gouty patients older than fifty show less than half of them able to concentrate urine to a specific gravity greater than 1.020 (Table 8). More precise tests for renal function show a smaller percentage of persons over fifty within the range for normal. We have seen few patients with gout older than fifty who have renal function that is normal by all of the tests enumerated above. It was apparent from the several measurements that the ability to form normal amounts of glomerular filtrate decreases simultaneously with the ability to concentrate solids maximally. Decreased excretion of phenolsulfonphthalein dye and impaired visualization of the kidneys by x-ray examination after injection of diodrast occur next. Increase of serum nonprotein nitrogen is a late manifestation. Most of the patients who show a persistent elevation of nonprotein nitrogen die within two or three years after the elevation has been noticed first. Urate calculi may be considered a complication also. These are found in an

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with malignant hypertension but is not increased in patients with hypertension associated with gout.²

Premature Arteriosclerosis Premature development of microscopic sclerosis of the larger arteries of the body has been observed in patients with gout. Some of our patients in the third and fourth decade of life have shown sclerosis of the large vessels by x ray examination. This is presumed to be premature sclerosis. It is doubtful, however, whether the sequelae of arteriosclerosis are more frequent in patients with gout older than fifty than in the similar group of patients afflicted with other chronic degenerative diseases.

Articular Complications Such complications of gout rarely are serious and almost never are fatal. Although urate deposits may be widely distributed throughout the joints of the body, gouty patients rarely become chronic invalids from gouty arthritis *per se*. Several patients in our series were invalids temporarily but following medical and surgical treatment they resumed an ambulatory status. A patient should not be considered a cripple from gouty arthritis until full advantage has been taken of medical and surgical treatment.

TREATMENT

There is no cure for gout, although various measures have been proposed which purport to control symptoms and maintain the patient in a relatively satisfactory condition. It may not be surprising that a cure for articular gout has not been discovered but it is a disappointment to students of the disease that methods have not been devised to reduce the urate content of the body. Metabolic gout appears superficially to be a relatively simple dysfunction. It is believed by us that all that would be needed would be a means to produce a permanent reduction of the concentration of urate in body fluids. If the urate concentration could be maintained permanently in the normal range, gouty arthritis should never develop. Although no cure for metabolic gout has been devised, skillful treatment of gouty arthritis may alleviate symptoms as effectively as modern treatment alleviates symptoms of diabetes mellitus.

The Acute Attack Acute gouty arthritis may best be treated by supportive measures as well as by specific ones. Bed rest and a soft diet with

symptoms. No sincere claim has ever been made that any of the supporting measures significantly influence the course of an acute attack or exert any permanent retardation upon the development of chronic deforming changes.

Colchicine, on the other hand, does influence the course of the acute attack, and there is some evidence that suggests that it may postpone the development of chronic deforming changes.⁷ Patients who are known to have gout should not be without a vial of colchicine tablets at any time. We do not advise the wine or tincture of colchicine because of the unreliable strength of either preparation. The administration of colchicine at the earliest possible moment after the onset of acute symptoms is most important. Every hour lost during the early stages of the acute attack may entail unnecessary suffering subsequently. Ingestion of colchicine should begin with the onset of acute symptoms, however mild. One 1/120 grain tablet should be taken every one or one and a half hours and continued without interruption irrespective of the time of day or night. During a mild attack joint symptoms

will subside after four or five tablets have been ingested. In a moderate or severe attack, tablets should be taken until the onset of gastrointestinal symptoms. This usually requires from twelve to sixteen tablets. The appearance of nausea, vomiting, and diarrhea is the signal to stop colchicine as well as to start the administration of a gastrointestinal sedative such as camphorated tincture of opium. The prolonged ingestion of colchicine, sufficient to produce gastrointestinal symptoms, is necessary for the treatment of a severe attack. Nothing is gained, however, by unnecessary prolongation of these untoward symptoms. Relief of joint distress should begin within twelve hours after colchicine administration. The above described regimen has been identified as a full course of colchicine and it should not be repeated until at least two days have elapsed, rarely is it necessary to repeat the course until a subsequent attack. If the acute attack has not been diagnosed early, or treatment has not been instituted early, it may be necessary to repeat the course.

Treatment of Chronic Gout and Prevention of Acute Exacerbations. Treatment of this phase of the disease is less satisfactory but is equally important. Unequivocal evidence is not yet forthcoming which explains the variation in severity of the disease among gouty patients. It is our belief, however, that the more frequent and the more severe the acute attacks, the greater the likelihood that the patient will develop chronic deforming joint changes. Therefore, any regimen which diminishes the frequency or aborts the severity of an acute attack may accomplish as much toward prevention of joint changes, subsequently, as it does toward sparing the victim hours or days of pain during the acute exacerbation.

It is essential to avoid physical strain or a direct blow on a joint, an acute infection, exposure to cold or dampness, surgical operations, or over-indulgence in food and drink. The prophylactic use of colchicine is indicated if any of these events are anticipated or develop unexpectedly. The first sign of an impending upper respiratory infection should be the warning signal to take a few colchicine tablets. Prophylactic use of colchicine before a holiday meal may not be amiss. Similarly, the use of colchicine before and after a surgical operation is to be recommended. In support of this statement, the incidence of postoperative gout has been reduced in our series from 86 per cent before the regular use of colchicine to 8 per cent.⁸

Asymptomatic Periods. The use of colchicine during asymptomatic periods as well as during acute attacks is recommended.⁷ Patients who have an attack of gouty arthritis occasionally may take from three to six tablets a week, others may take two tablets on three or four days each week. Severely afflicted patients should take from one to three tablets each day in the year. Since this practice has been instituted more than ten years ago, we are con-


support of most of the claims are lacking. Once a drug or a diet has been handed down from textbook to textbook, thoughts become accepted as facts and skeptics are branded as heretics. A low purine intake and abstinence from alcoholic beverages have been responsible for many unhappy patients. These measures are routinely prescribed by many physicians for patients with

gout because they are so recommended in many textbooks of medicine. They have been recommended empirically in most instances and lack controlled observations.

In an attempt to collect experimental data on this subject we have noted the same frequency of attacks in gouty patients on a moderate purine intake as on a scrupulously low purine intake. Obviously, foodstuffs such as liver, kidneys and sweetbreads which are high in purines are to be avoided. A moderate portion of red meat, however, is allowed each day. In addition to the provision for a balanced diet and an adequate protein intake, meat is advocated because it has been shown that proteins have a diuretic action upon purine substances. None of our patients is forbidden alcoholic beverages, temperance in all things is the advice that is dispensed. A few patients are convinced that even a small quantity of a particular kind of alcoholic beverage has been responsible for an acute attack. Under such circumstances, the patient is advised to avoid the offender. The privilege of living an essentially normal life is appreciated by most patients and the physician is usually rewarded by full cooperation at all times. A large urine output is desirable and can be achieved only by a liberal fluid intake. Since the solubility coefficient of urate in body fluids is low, the greater the urine output, the greater the available fluid to carry off urate. Since there is a limit to which urine output will enhance urate excretion, the patient need not suffer from induced nocturia, polyuria during the day is sufficient.

Surgical Treatment The indications for surgical treatment are (1) Large subcutaneous tophi which have become unsightly and require removal for cosmetic reasons rather than because of symptoms which they produce. Small tophi of the hands and feet which interfere with the wearing of gloves or shoes belong in this group. (2) Painful tophi of exposed areas of the body, such as those which involve the olecranon bursae, knuckles, terminal phalanges of the fingers, toes and heels, respectively. (3) Deposits which interfere with the movement of the fingers and hands from involvement of extensor or flexor tendons. (4) Any discharging sinus associated with a tophaceous deposit, and (5) extensive osseous involvement of the fingers or toes. The successful operative results are attributed by Linton⁸ to the adherence to certain rules. The incidence of arteriosclerosis among gouty patients is an important factor. Great care must be exercised in order to maintain an adequate blood supply, particularly in the lower extremities and to prevent trauma to the parchment thin skin which overlies many tophi. Arterial tourniquets are contraindicated. Drainage is unnecessary and wounds heal exceptionally well. Postoperative sepsis is not a common complication. There should be no hesitation in amputating anklyosed toes. This has been done in several instances in our series. The patients have been rehabilitated and with properly designed shoes, their gait has been restored and they have been able to walk in an essentially normal fashion. The end results justify radical procedures. If a tophaceous deposit involves an important tendon, it is considered desirable to remove the tophus, leaving as much as possible of the tendon. In instances where it is impossible to recognize the tendon because of extensive tophaceous deposits, tendon remnants may be sacrificed with a surprisingly satisfactory postoperative result.

Miscellaneous Drugs Cinchophen has enjoyed a reputation as a specific for gout and is prescribed extensively.³ Its use, however, has been disap-

proved by the Food and Drug Administration of the United States Government because it is a potential poison. We are heartily in accord with this ruling. Acute yellow atrophy of the liver is the most serious consequence of the use of cinchophen. Furthermore, we have never seen convincing data to show that cinchophen is indispensable in the treatment of gout. Except for the diuretic action upon urate, cinchophen possesses no known restorative properties affecting the disordered purine metabolism. The analgesic effect upon *joint discomfort is not specific and may be achieved as effectively in gouty arthritis by the use of colchicine combined with salicylates.* Hence, the credit attributed to cinchophen as a specific for gout seems unwarranted. Salicylates have a diuretic effect upon urates similar to cinchophen and are not endowed with as serious toxic properties. Hence, if any urate excreting substance is desired in the treatment of gout, salicylates are to be preferred. From 2 to 4 gm. of sodium salicylate may be taken daily during the acute attacks and for two or three days each week in the intercritical period. Since the effect upon urate excretion is lost after a short course of salicylates, this substance should be taken intermittently and not continuously. 

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CHAPTER 16

DYSFUNCTIONS OF THE ENDOCRINE GLANDS

NATHAN S. DAVIS, III

PATHOLOGIC ANATOMY

It is not yet possible to differentiate the normal involutionary anatomic changes in the pituitary, thyroid, and adrenal glands and the testes from the pathologic degenerative changes that have developed as a result of damage due to mild or severe, acute or chronic disease or nutritional deficiency, to the ingestion, in sufficient amounts, of metals, pharmaceuticals and toxic chemical compounds which interfere with normal cellular chemistry, to the effects of abnormal, hyper- or hypofunction of one or more of the endocrine glands on the others and on metabolism in general, to the inability of the liver and other tissues to normally catabolize hormones, other products of tissue metabolism or toxic endogenous or exogenous compounds (See Chapter 3)

The pathologic anatomy commonly found in these glands in the aged closely resembles that seen in younger individuals who have been starved, who have had chronic debilitating diseases, or have had one or more of these glands extirpated¹ Atrophic changes predominate but hyperplastic ones may also develop The amount of change, which differs from one endocrine gland to another, depends upon the particular etiologic factors to which the individual has been subjected throughout life their magnitude or intensity and duration

In the aged, the testes, the pituitary, thyroid, and adrenal glands are usually decreased in size This decrease is relatively more pronounced in the pituitary and thyroid though diffuse or adenomatous hyperplasia is found not infrequently in these glands, in the adrenal cortex, and rarely in its medulla^{2 3 4} The reduction in the size of the glands is due to a decrease in the size and number of cells, which is only partially compensated for by an increase in the thickness of individual collagen fibrils and in the mass of the stroma These changes seem to be caused by transfer of water from the cells to the extracellular spaces where it induces connective tissue changes similar to those found in chronic cardiac edema¹ (See p 390)

Since diffuse or adenomatous hyperplasia of the thyroid seems to be due to a deficient intake of iodine that in the pituitary and adrenal glands may also be due to a specific, but as yet unidentified, type of nutritional failure It is also possible that when subjected to excessive stimulation by trophic hormones, hyperplastic but nutritionally deficient tissue may produce toxic products as do toxic goiters, and when overstimulated by anabolic or trophic hormones, the hyperplastic tissues may undergo malignant degeneration

In addition to these nonspecific changes, there is generally a reduction in the iodine and colloid of the thyroid in old age Alterations develop in the chromophile cells of the anterior pituitary similar to those found in inactive

or fatigued mature glands and following castration or thyroidectomy. These are a reduction in the number of eosinophile cells (which secrete the growth, thyrotropic, and lactogenic hormones) and, to a lesser extent, in the basophile cells (which secrete the adrenotropic and gonadotropic hormones). Some of the cells of the anterior lobe may migrate to other portions of the gland. The senile changes found in the adrenals of the aged are as a rule most marked in the inner layer of the cortex, less in its outer layer, and least in the medulla. The hormone-producing portion of the testis, the interstitial cells of Leydig, usually atrophies before the production of normal or abnormal spermatazoa ceases, possibly because the resulting hypofunction stimulates the release of the follicle stimulating pituitary hormone. The basement membranes of the testicular tubules become thicker and the spermatogonia are so altered that they resemble the small round cells of immature testis. There is also an increased pigmentation of the interstitial cells in the aged testis.⁵

PATHOLOGIC PHYSIOLOGY

In summing up his views on the nature of the mechanism of aging, Warthin concluded that age was due to a loss of growth energy which might result from an increase in specific growth inhibiting factors, a decrease in specific growth stimulating factors, or a combination of these two factors acting conjointly.⁶ Nutritional failure may cause the development of either or both of these factors and of changes in the endocrine glands at the molecular, cellular, and organ levels in the aged. Such failure may be due to any one or any combination of the following conditions:

- 1 Deficient intake of essential nutriment
- 2 Deficient absorption or assimilation of essential nutriment
- 3 Diminished blood supply due to sclerotic changes in the cardiovascular system
- 4 Inhibition of the formation or action of the cellular respiratory enzymes by the products of bacterial growth, by the ingestion of antivitamin, minerals and various toxic chemical compounds having such effects, or by allergic reactions
- 5 Excessive demands for nutritional elements essential to the normal function of a particular organ or tissue which has been made hyperactive because of neurogenic, hormonal, or other chemical stimuli, and which may cause what is usually an adequate intake to be deficient
- 6 Exhaustion of the liver and other tissues so that they can no longer catabolize certain hormones which, as a result, accumulate in the system and produce changes similar to those produced by their hypersecretion or by a decreased excretion of antagonistic hormones
- 7 Alterations in the secretion of hormones having antagonistic effects and of antihormones
- 8 Inability of aged tissues to respond to nutriment and hormones in a normal manner

The exact nutritional requirements of the endocrine glands will not be known until the specific causes of the reversible and irreversible changes at the cellular and organ levels and the different effects produced by exposures to the same causative factor in varying amounts and for varying periods of time have been determined.⁷ Much remains to be learned also about the

effects of hormones on general metabolic processes, on the activities of the other endocrine glands, on the action of other hormones and on enzymes. It is known that some hormones have effects which are anabolic, others catabolic, some stimulating, others inhibiting, that some are synergistic, but others antagonistic. Degenerative or involutionary changes in one part of the endocrine system may cause atrophy and hypofunction in some, hyperplasia and hyperfunction in other portions, and this may modify the rate of development and character of geriatric pathology.

It is known that all of the vitamins are relatively concentrated in the endocrine glands, that the ascorbic acid content of the adrenals is highest, of the pituitary next, that arginine, tryptophane, and Vitamins A, D, and E are required for normal spermatogenesis, that Vitamin B complex is required for the normal function of all,^{8, 9} that tyrosine is a component of some of the pituitary hormones of thyroxine, epinephrine, and renin, that the functional activity of some of these hormones may be influenced by the particular isomer used in their anabolism,¹⁰ that 1 mg. of iodine daily seems to be required for normal thyroid activity. Just as iodine deficiency seems to be associated with diffuse or adenomatous hyperplasia of the thyroid, the specific deficiency of an essential nutriment may be the cause of the adrenal and pituitary hyperplasia of the aged. Administration of the corticotropic hormone has been shown to reduce the ascorbic acid and cholesterol content and to cause hyperplasia of the adrenal cortex.¹¹ Thiamine deficiency also causes cortical hyperplasia, but pantothenic acid deficiency causes cortical hemorrhagic necrosis.¹²

Posterior Pituitary Hormones Two important hormones, one a vasopressor substance, called pitressin, the other, an oxytocic substance, called oxytocin, are formed by the neural posterior lobe of the pituitary gland. The uterine muscular contractions stimulated by oxytocin are increased when estrogens have previously been administered. This effect is antagonized by progesterone.

The vasopressor effect of pitressin is not abolished by denervation of the blood vessels. While it stimulates smooth muscle contraction in the gall bladder, intestine, ureters, and urinary bladder, its most important action is the stimulation of water reabsorption from the renal tubules. This causes pitressin to have a marked and rather prolonged antidiuretic effect. Hypofunction of the posterior lobe of the pituitary causes diabetes insipidus, the polyuria of which is relieved by the administration of pitressin. Its formation is stimulated in dehydration by processes involving nerve reflexes and chemical reactions.

Intermediate Pituitary Hormones This portion of the pituitary gland secretes a hormone, intermedin, which influences the dispersion of the epidermal melanophores, and a metabolic factor which elevates the basal metabolic rate and lowers the respiratory quotient in thyroidectomized animals.

Anterior Pituitary Hormones Because of its multiple products which influence general metabolism as well as the activity of other glands, the anterior lobe has been termed the master endocrine gland, though its functions may be modified by other endocrine dysfunctions. Its four types of cells secrete six known hormones which probably are responsible for most of the activities which have been ascribed to many crude products extracted

by various methods. The six hormones are the lactogenic, growth-stimulating, follicle-stimulating, luteinizing, thyrotropic, and corticotropic hormones. They are proteins which can be digested and inactivated by the gastrointestinal enzymes. Extracts of the gland have been shown to have diabetogenic, glycostatic, glycotropic, and ketogenic properties. The acidophile cells of the anterior pituitary secrete the growth, lactogenic, and thyrotropic hormones.⁸ The luteinizing hormone was formerly thought to be secreted by these cells because changes induced by castration resemble closely those caused by thyroidectomy.⁶ The basophile cells secrete the corticotropic or adrenotropic, the follicle stimulating, and luteinizing hormones. It is possible that the eosinophile cells form a rudimentary gonadotropin which is transformed into the follicle-stimulating and luteinizing hormones by the basophile cells. The chromophobe cells are believed to have no secretory function though tumors composed of these cells may cause hypogonadal syndromes.

Pituitary tumors may cause either hyperfunction or hypofunction of this portion of the gland. Simmonds' disease develops as a result of general hypoactivity of the anterior lobe. Its symptoms resemble those of accelerated aging. Hypofunction of the anterior lobe, which may be due to disease, inanition, or severe vitamin deficiency, causes anorexia, atony of the gastrointestinal tract, lowered basal metabolism, hypoglycemia, subnormal temperature, low blood pressure, bradycardia, weight loss, amenorrhea, impotence, loss of axillary and pubic hair, hypothyroidism, anemia, weakness, mental apathy and coma. This general pituitary hypofunction results in atrophy and hypofunction of the adrenal cortex, liver, kidneys, gonads, pancreas, uterus, and thyroid.

Hyposecretion of the growth hormone during development causes dwarfism, infantilism, and obesity. Its hypersecretion during prepuberal growth causes gigantism and, in adults, acromegaly. In males the follicle stimulating hormone stimulates spermatogenesis, and the luteinizing hormone the production of the male sex hormone. The secretion of the gonadotropic hormones is affected by nervous stimulation and by their blood levels. The sex hormones inhibit the secretion of gonadotropic hormones, the pituitary concentration of which is increased following castration or the menopause. Hyposecretion of the gonadotropic hormones may cause obesity, mental deficiency, subnormal development of the genitalia and of secondary sex characteristics, feminism in males, disordered menstruation, and subnormal temperatures and basal metabolic rates.

The thyrotropic hormone, formed in the acidophile cells of the anterior

hyperplasia. The thyrotropic hormone is inactive when there is extensive thyroid atrophy. Repeated injections of this hormone cause antithyrotropic substances to be formed.

The corticotropic hormone, secreted by the basophile cells of the anterior

lobe, acts on the adrenal cortex to maintain its normal structure and function. Its hyposecretion leads to atrophy, its hypersecretion to diffuse or adenomatous hyperplasia of the adrenal cortex. It may cause an increase in pituitary basophilism or secretion in Simmonds'.

Androgens Testosterone is the male sex hormone secreted by the testicular interstitial cells of Leydig under the stimulating influence of the luteinizing hormone of the anterior pituitary lobe. Other male sex hormones are adrenosterone, a constituent of the adrenal cortex, and androsterone and dehydroandrosterone, both of which are androgenic excretory products. Only traces of testosterone are found before puberty, but thereafter it is secreted continuously in primates. The other androgens and the estrogens are found at all ages in both men and women. Before puberty and during old age the estrogens and androgens are about equal in amount, but the androgens are more abundant in the sexually active male and the estrogens throughout the reproductive period in the female. Their excretion is low in hypothyroidism, malnutrition, anemia, infections, and in hepatic diseases which prevent their destruction by the liver and cause their blood levels to rise. The androgens stimulate the development of secondary sex organs and characteristics. Testosterone is antagonistic to the estrogens. When administered in large doses, the male sex hormone depresses secretion of the pituitary follicle stimulating hormone to inhibit spermatogenesis. Spermatozoa usually continue to be formed for some time after age has caused hypofunction of the interstitial cells and a diminished testosterone secretion. However, the spermatozoa formed during old age are not normally active in most cases.

The effects of the androgens on secondary sex organs and characteristics and other changes caused by hypo- and hypergonadal activity are so well known that they need not be enumerated. However, since the testicular male sex hormone was identified as testosterone and has been synthesized, it has been shown to have other functions which are of greater importance to the maintenance of health in the aged than are those associated with sexual activity.¹³ Testosterone diminishes the urinary excretion of inorganic phosphorus, nitrogen, sodium, chlorides, and water but does not cause the reciprocal rise in ammonia excretion produced by the corticosterones, which also do not limit the urinary excretion of phosphorus, potassium, and nitrate. It seems to have an anabolic function associated with the development and maintenance of the supporting matrix of skeletal muscles, bones, etc. The hypogonadism of advancing years causes muscular atrophy and an osteoporosis in which there is a lack of matrix formation but no failure in mineralization such as occurs in *osteomalacia*.¹³ Albright and Reifenstein found testosterone more potent than estrogens and a combination of estrogens and androgens still more effective in the treatment of osteoporosis, which may be one of the first definite signs of hypogonadism to develop.¹³ The synthesis of radioactive testosterone¹⁴ should result in rapid advances in our knowledge of its functions during growth, maturity, and old age, of its activities as a carcinogen and in connection with hypertension.

The administration of testosterone does not stimulate spermatogenesis, indeed it may inhibit it. Thus, testosterone is of little, if any, value in overcoming the effects of aging, on sexual activity⁵ and in preventing or overcoming prostatic enlargement.²² It has carcinogenic effects in the male and anti

carcinogenic in the female, effects which are antagonistic to those of the estrogens. Castration and estrogen therapy have been found to be of value in treating prostatic carcinoma,²² decidedly retarding the rate of growth.

Adrenal Glands. Cortical Hormones The hormones and other products of the adrenal cortex affect the permeability of membranes, gluconeogenesis and the deposition of glycogen in the liver, renal function, somatic growth, atrophy of the thymus, pigmentation, and resistance to stress such as may be produced by hyperinsulinism, tetany, toxins, infection, and exposure to extreme cold. The corticotropic hormone of the anterior pituitary is required for the normal activity of the cortical cells. Desoxycorticosterone, which primarily affects the distribution and elimination of inorganic ions, especially sodium and chlorine, is formed in the outer layers of the cortex. Corticosterone, which affects carbohydrate and other metabolic activities, is formed in the inner layers of cells. Dehydrocorticosterone, dehydro-hydroxycorticosterone, and adrenosterone are other hormones which have been identified as cortical in origin. As the hypophysisoprivic animal secretes only an immature cortical hormone, the corticotropic hormone seems to participate in the transformation of products of the outer cortical layers into those of the inner, by the addition of an atom of oxygen to carbon 11 and the removal of a side chain.

Ascorbic acid deficiency causes adrenal atrophy, thiamine deficiency, adrenal hypertrophy, and pantothenic acid deficiency, hemorrhagic necrosis and rapid exhaustion of cortical lipoids in experimental animals.^{8, 15} Purpura fulminans, which follows hemorrhagic necrosis of the adrenal cortex due to the acute infectious diseases of childhood, is characterized by marked purpura, hypoglycemia, and azotemia. Chronic adrenocortical insufficiency causes the progressive weight loss, fatigability, melanin pigmentation, hypochlorhydria, anorexia, nausea, vomiting, hypotension, and anemia of Addison's disease. Exercise, infection, the administration of potassium, or withdrawal of sodium salts causes "Addisonian crises" with shock, dehydration, azotemia, and retention of inorganic phosphate and sulfate to develop in this disease. Its hyperfunction may cause precocious puberty and adrenal virilism in females, pronounced masculinization and pseudohermaphroditism in early life, isosexual precocious maturity and hirsutism in late prepuberal life, hirsutism and feminization in adult males, and sometimes hypertension in the adults of either sex. Adrenal cortical function is essential to life.

Adrenal Medulla Epinephrine, which is the hormone produced by the modified sympathetic ganglion cells of the adrenal medulla, is not essential to life. It is a sympathomimetic hormone the secretion of which is controlled by the preganglionic cholinergic fibers, which are active during sympathetic stimulation due to fear, rage, pain, asphyxia, anesthesia, hemorrhage, strenuous substance,

return to normal blood pressure levels. It relieves bronchiolar spasm. Pulmonary arterioles are relatively resistant to its vasoconstrictor effects. It causes coronary vasodilatation to sustain the heart which must work harder when the blood pressure is elevated. It does not cause vasoconstriction in working skeletal muscle.

The administration or hypersecretion of epinephrine --
 levels of sugar and lactic acid by --
 glycogen --

by in
 restor
 adren.
 cause
 extensive resorption of bone, and hyperglycemia because of hyperfunction of the medullary cells

Hypertensive cardiovascular renal disease is not thought to be caused by adrenal medullary hyperfunction but has been etiologically connected with adrenal cortical activity in pituitary basophilism --
 syndromes ¹⁶ (See p 467) Yet the

restrictive the --
 of large
 not infrec

cortex, as
 hyperfunc. may frequently be involved in its causation. It is known that high blood levels of normal or abnormal corticosterones may inhibit renal deamination by oxidation and stimulate deamination by decarboxylation, the formation of pressor amines and possibly renin or a renin-like pressor substance ¹⁷. Reports that prolonged and severe shock exhausts the adrenal glands, inhibits the secretion of epinephrine, and activates an auxiliary renal pressor mechanism, make it seem possible that essential hypertension may at times be an indirect result of medullary hypofunction. Of course, hepatic dysfunction which interferes with the normal catabolism of epinephrine and androgens may cause a rise in blood pressure.

Thyroid Hormone. The hormone --
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by acting as a catalyst or by increasing the amount, potency, and effectiveness of the cellular respiratory enzymes. During the growth period, it increases the size of cells but not their number, and so differs from the pituitary growth hormone with which it has in other respects a synergistic action. It influences salt, protein, and cholesterol metabolism. Thyroid hypofunction causes an accumulation of protein in the extracellular spaces, a decreased rate of exchange and urinary excretion of calcium and phosphorus and other inorganic salts, and retention of water. The hypercholesterolemia due to hypofunction of the aging thyroid may contribute to the formation of atheroma, and the associated defect in calcium metabolism to calcifications. Hypothyroidism lowers the basal metabolic rate.

Hyperthyroidism increases the basal metabolic rate --
 stores and, by damaging liver cells --
 tional instability --
 increased
 muscular
 cardia, elev
 blood flow

myasthenia and
 tolerance for creatin, tachy-
 of the blood pressure, and increased

SYNDROMES IN THE AGED DUE TO ENDOCRINE DYSFUNCTION

As thyroid, adrenal, or gonadal hypofunction stimulates the secretion of trophic hormones by the pituitary, it is understandable that Raab stressed the resemblance between syndromes of old age and Cushing's disease, characterized by pituitary hyperfunction, involving especially the basophile cells, and by adrenal cortical changes,¹⁸ while others stress their resemblance to

lation by trophic hormones or other factors which normally increase their activity, the clinical picture of aging usually resembles syndromes due to polyglandular endocrine dysfunction and presents features of abnormal as well as of hypo- and hyperfunction. Furthermore, the general metabolic changes characteristic of the nutritional failure and the vascular changes of old age modify the symptoms and findings to mask the picture and make difficult the diagnosis of endocrine disease. For example, some of the aged, with diffuse or adenomatous hyperplasia of the thyroid, who develop "toxic goiters" have only a moderate rise in pulse and metabolic rates, while others develop auricular fibrillation or more or less anginal or congestive heart failure. Thyrotoxicosis causes less weight loss but greater weakness in the aged, some of whom become nervous and others apathetic. In geriatrics "toxic goiter" is usually associated with adenomatous hyperplasia, and is relatively more common in males.¹⁹ As such patients are not good surgical risks,¹⁹ therapy with one of the thiouracil derivatives seems to be indicated. Such preparations must, however, be administered with great caution as average doses may cause excessive reduction in thyroid function or marked depression of the hypofunctioning hemopoietic system in the aged.

As the senescent individual with symptoms suggestive of hypothyroidism does not present evidence of the increased secretion of the thyrotropic hormone which develops following thyroidectomy in young people, it seems that the "toxic goiter" in the aged may develop when the hypofunctioning thyroid is overstimulated by the thyrotropic hormone of a pituitary in which there has been less degenerative and involutional change. Similarly, the dysfunctions of other endocrine glands may depend on hypofunction or hyperfunction of other parts of the system with the clinical pic-

secretion of the male sex hormone stimulates the pituitary to increase its output of both the gonadotropic and corticotropic hormones, and causes the formation of excessive quantities of steroids some of which may be

formation of pressor amines and renin or a renin-like pressor substance, such pluriglandular dysfunction may cause the elevated blood pressure frequently

found in the aged. It must be remembered, however, that there is a variety of hypertension in the aged characterized by a high systolic and normal or low diastolic pressure which is caused by the increased capacity of the inelastic, dilated, and elongated larger arteries, not by vasospasm or arterio sclerosis with narrowing of the lumina of the arterioles and increased peripheral resistance.

THErapy IN THE AGED

Therapy of the syndromes in the aged due to hyperfunction or altered secretion of toxic products by glands has not been as successful as in younger individuals. This is due to the fact that the aged are not good surgical risks, to the changes in function that follow surgical removal, radiation, or effective drug therapy, which magnify or distort those changes caused by the disordered function of other parts of the endocrine system, and to lack of effective therapeutic measures.

Treatment of endocrine hypofunction also has not been effective in the aged. Replacement therapy with one hormone usually is not satisfactory because of the resulting altered secretion of other hormones and the inability of tissues to catabolize hormones. Aged tissues are less responsive to hormonal action than younger tissues. As the disorders of old age caused by altered endocrine function are pluriglandular in origin, they should benefit most by the administration of the several hormones required to restore normal function in the particular patient. Korenchevsky and Jones have shown that in ovariectomized rats, the administration of androsterone, estradiol benzoate butyrate, and thyroid hormone in certain combinations is much more effective than any one of them alone in minimizing the changes in various organs which follow this procedure.²⁰ A combination of androgens and estrogens has been reported to be more effective than either of these hormones alone in the treatment of senile osteoporosis.¹³ As the metabolic changes resulting from senile testicular hypofunction are not qualitatively or quantitatively identical with those following castration, the same type of pluriglandular therapy would not be effective even if the quantities administered were proportionately reduced. To make effective replacement therapy

in the aged, a more precise knowledge of the changes in the endocrine system with age would be required—one dependent on the amount of involutional plus degenerative changes involving each of the glands, on the resulting alterations in function of the endocrine system as a whole, and on their effects on general metabolism. Until it is possible to estimate fairly accurately the amount of altered function in each endocrine gland of a particular individual and to determine the amounts and proportions of the hormones required to compensate for such deficiency, even pluriglandular therapy will not be effective. Some such scientific basis for the administration of hormones, either singly or in combination, must be developed before their widespread use in treating the aged can be advocated.

Despite the obvious need for more precise knowledge and methods of evaluating endocrine activity, there are a few rather dogmatic statements which can be made regarding the administration of pituitary, thyroid, adrenal, and testicular hormones, singly or in combination.

1. Aged endocrine and other tissues do not respond in the same manner

as younger tissues to the pituitary trophic and growth hormones or to thyroxine

- 2 Adrenal cortical hormones are less effective in the treatment of senile hypofunction than they are in growing and mature individuals
- 3 The administration of testosterone will not rejuvenate aged men or restore their sexual activity⁵ It may inhibit spermatogenesis by lessening the secretion of the follicle stimulating pituitary hormone and may stimulate hyperplastic tissue to undergo malignant degeneration The male sex hormone does not appreciably influence benign prostatic hypertrophy^{5, 21 22 23}
- 4 The administration of testosterone is indicated in the treatment of the so-called "male menopause" *only* when it can be proven that there is a low blood testosterone level Most men presenting this syndrome have normal blood androgen levels and their symptoms are not caused by testicular hypofunction, but by nervous fatigue, malnutrition, or disease^{21 23 24} The reported benefits from testosterone therapy of vascular disease are probably due to its effects on protein, mineral, and general metabolism²²
- 5 Proper mixtures and dosages of hormones will accomplish more in overcoming the general metabolic changes due to hypogonadism or other endocrine hypofunction in the aged than they will in restoring specific functions that have been lost Such treatment will be more effective if combined with a diet that is adequate in all respects If there has been more or less nutritional deficiency, especially if it has been slight and of long standing, relatively large doses of the essential elements that have been lacking must be administered for a long period of time if reversible involutional and degenerative changes are to be overcome and functional efficiency improved Of course every effort must also be made to remove all possible causes of nutritional failure

Because irreversible changes cannot be corrected, and may make it impossible to restore function adequately, the involutional and degenerative changes of old age tend to progress Therefore, the earlier the treatment directed at the elimination of the causes of nutritional failure and the restoration of normal function at the molecular, cell, and organ levels is begun, the less will be the irreversible changes (See Chapter 5) Such early treatment will postpone or arrest the development of the pathology characteristic of old age and minimize the effects of nutritional failure on endocrine and general metabolism, will make it possible for the aged to be hale, hearty, able to work, and to enjoy ripe old age

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SECTION III

DISORDERS OF THE MIND AND NERVOUS SYSTEM

CHAPTER 17

MENTAL DISEASE

WINFRED OVERHOLSER

IN any discussion of mental disorders, a few words concerning the present-day conception of what we refer to as "mind and mental functioning" are in order. The old notion of the mind as a sort of homunculus seated perhaps in the region of the pineal body, quite independent of the physical organism but directing its activities, has during the present century given way to what may be termed a holistic concept. Thanks to the work of Freud, of Meyer, and of William Alanson White, to mention only a few of the significant contributors to the field of psychiatry, we find today a general recognition of the fact that what we call mind is but one aspect of the functioning of the physical organism, that it is an abstraction which summarizes the manner in which the individual as a whole reacts to his environment, that environment being constituted of his physical body, his previous experiences, the climatic and other physical conditions surrounding it, and the conduct and attitude of his fellow men.

The manner in which anyone will react to a situation is dependent on a variety of factors. It is conditioned in part by his bodily structure, by his degree of intelligence, by the circumstances among which he was reared, by his contacts with his past, his siblings and his playmates, his education, the impact of tradition upon him—in short, the social milieu—and that multiplicity of economic and social facts of which we are conscious. The manner in which his physical organism and the various parts thereof function is an important factor in the manner in which any given set of circumstances will cause him to react. The proverbial optimism of the tuberculous patient, the querulous attitude of the dyspeptic, have passed into common speech and are widely recognized. Likewise, through the development of psychosomatic medicine, thanks to the researches of Cannon, Dunbar, Weiss and English, Wolff and others, we are coming to a realization that the emotions play a large part in the functioning of various organs of the body, and vice versa. There is thus a close and intimate interrelation between the body and the mind. In the past internists and surgeons have been somewhat oblivious of this fact, and in the same manner the psychiatrist has sometimes erred in paying too little attention to the physical findings in his patients. Today, however, we find the psychiatrist, the internist, and the surgeon, as well as the various specialists, meeting on a common ground and recognizing that they are all dealing with the human being and that that human being must be considered *in toto* rather than as an assemblage of parts.

MENTAL HYGIENE OF OLD AGE

The entire process of living is one of adjustment. This is true whether we look at the physical side and consider such homeostatic mechanisms as the maintenance of bodily temperature or whether we consider the adjustability of the individual as a whole, and his ability to meet various social situations. Here again the adjustability of the physical organism cannot be divorced from that of the personality, and we shall see as we proceed in the present chapter how, with the loss of physical elasticity, certain failures of adjustment to various social and economic stresses and strains likewise manifest themselves. One truth which recent studies in the field of geriatrics have demonstrated conclusively is that aging is not a mere question of the calendar, indeed, the body does not all age together. In some persons the process of senescence of some organs begins astonishingly early, and in others it seems to be postponed almost indefinitely. Some individuals remain young until late in life, whereas others seem to be old although by the calendar they are hardly in their maturity. Again, recent studies have indicated that in the past altogether too much stress has been laid upon the organic factors in the mental disorders of advanced years and not enough on what may be termed the psychologic hazards of aging. Some patients who have shown profound mental changes attributable to advancing years have been found at autopsy to show but little pathologic change of the brain, whereas, on the other hand, others in whom striking cerebral lesions can be demonstrated have shown an extraordinarily good ability to adjust and to meet the demands of their environment. Far more important than the organic changes is the functioning of the various organs, and even more so the functioning of the personality as a whole. The patient with the well integrated personality, the sound individual who has an ability to adjust to changing situations, may survive intact or substantially intact the ravages of cerebral arteriosclerosis, whereas the schizophrenic, the rigid, or the neurotic type of individual may, as loss of elasticity progresses, show lines of cleavage which may lead even to institutional care. The personality again tends to become less elastic and more brittle as senile changes progress in the physical organism.

Onset of Senescence. Senescence does not occur overnight. It is an extremely gradual, not to say an insidious, process, and its exact onset can rarely be dated. Two of the most definite milestones may be said to be the loss of accommodative power in the eye so that bifocal glasses become necessary, and the development of menopausal symptoms together (in the male) with decrease of sexual vigor. There are definite climacteric symptoms in the male, but they tend to occur somewhat later than in the female. At the same time there is observed a loss in reserve power—the patient becomes more easily dyspneic, the circulation is not so adequate for unusual exertion, some changes may be noted in the arteries, and the blood pressure may show a tendency to increase. These changes in the functioning of the various organs of the body certainly must not be minimized. They are all factors in the development of what we know as old age, with all that that implies.

There are certain psychologic concomitants, however, which there is a tendency to overlook. The development of the early signs of flagging physical vigor is a warning to the patient that he has passed the zenith; that from then on the time is growing shorter in which he may accomplish his

life's ambition. This in itself tends to impress upon him the errors of judgment, the mistakes which he has made during his past life and which have resulted in his not having attained the position in his field which he has desired. At the same time he realizes too that he may soon be retired from his employment. This likelihood had become rather marked before the development of the industrial boom about 1941. Until that time many industries had made it a plan to retire many of their employees at about forty-five, and to hesitate to employ anyone over forty. Fortunately, many industries learned during the war that the older person has much to contribute in the line of sound judgment based on long experience, even though he may be slightly less agile than the younger worker. The specter of retirement or lack of employment, and of possible physical impairment with resulting accentuation of the inability to work, may hang constantly over the patient. There is thus worry over finances and over physical health.

At the same time the aging person finds that his circle of friends is being narrowed by death, as his contemporaries are dying off, he finds it increasingly difficult to recruit friends, so to speak, from the younger age group, with the result that he tends to feel himself more and more alone. At the same time the attitude of his children, particularly if he lives in the same household with them, may be one of increasing impatience and fretfulness, with a readiness to criticize and to direct, so that here again he may feel himself unwanted and very much alone. It is sometimes easy to see the basis of fact on which an elderly person develops the delusion that he is being robbed by his children or that his death is desired!

The part played by deafness in the development of feelings of suspiciousness and isolation has not been generally realized. The inability to keep up with conversations, and the resultant omission of the deaf person from the social intercourse of the family and friends, serve to emphasize a feeling of apartness and to cause the psychologically predisposed to suspect that the conversation is about him. The advance in the manufacture of hearing aids has done much to alleviate this particular burden of advancing years. (See Chapter 20.) A tendency is generally noted in older persons, especially in those of limited education, to undergo a narrowing of interests, in this manner the development of introspectiveness and querulousness is emphasized.

Approaches to Treatment. All too often in the past the internist when called upon to advise an aging person upon the condition of his arteries or his kidneys or prostate has been inclined to overlook, in laying down a regimen, what I have denominated above as the psychologic hazard. Much can be done to make the life of the aging person more comfortable and useful, and to enable him to expect a more varied adjustment to the demands of the situation if some of these simple principles of mental hygiene are borne in mind.

First of all, of course, comes the assistance to the patient in overcoming his physical difficulties or adjusting to them. This involves not only palliative or curative treatment of the disorder in question, but also the developing in the patient of a suitable mental attitude—a recognition of the fact that although he is somewhat handicapped, life is not completely at an end for him and that there are compensations. As a corollary, care should be taken to protect the aging person from injuries, an injury often seems to hasten

materially the aging process. Burdens upon the sense organs should likewise be avoided so far as possible.

Second is the necessity of conserving his intellectual interests, if he has any, or, failing that, assisting him in finding hobbies. Their value has been much underestimated; many persons have been inclined to look upon hobbies as a fad, whereas, as a matter of fact, they have a decided value in mental and physical hygiene. Preferably, of course, some useful work should be found for the patient to do, at least about the house. This will serve the purpose of making him feel that he has a place and that he is wanted. We are all familiar with the untoward effects of an abrupt withdrawal from the regular occupation. If, however, no useful work is available, the hobby is all the more important. With more leisure we may expect eventually a better system of education for the use of that leisure, something which is seriously lacking at present. Again, the necessity of companionship should be borne in mind, and the patient should be assisted to find friends. Perhaps the greatest single hazard of old age is the feeling of loneliness which I have mentioned earlier.

Finally, and not the least important, is enabling and encouraging the aging person's family to attempt to understand him and to be willing to make some adjustment to his peculiarities. The forgetfulness, the tendency to reminisce and to tell about the good old days, the tendency to ultraconservatism, and the feeling that the present generation is headed for damnation, are all parts of the picture of aging. If the family can only be encouraged, however, to have some patience with these peculiarities, much can be done to make the elderly person's life more comfortable and more useful. Such a seemingly minor detail as encouraging the forgetful older person to carry a scratch pad and use it for jotting down details will do much to reassure him and add to his self confidence. To those who are interested in these constructive aspects of the treatment of old age, a study of the clinic founded by Dr. Lillian J. Martin in San Francisco is recommended. Dr. Martin started this clinic when she was herself seventy-five years of age, and described its functioning in a volume entitled "Salvaging Old Age."⁵

INCIDENCE OF MENTAL DISORDERS

Before proceeding to a discussion of the types of mental disorder met with in the later age groups, a few words regarding statistics may be tolerated.

From 1910 to 1936 there was an increase in the admissions per 100,000 general population of persons aged 60 to 69 from 114 to 191, or 67½ per cent, and in the age group of 70 and over the increase was from 184 up to 418, or 181 per cent. Furthermore, the rate of admissions to mental hospitals on account of psychoses due to cerebral arteriosclerosis in the same period increased 536 per cent. A recent study of the Pennsylvania statistics by Johnson offers some prognostications which give food for serious thought to the mental hospital administrator.⁹

Dayton in his significant volume entitled "New Facts on Mental Disorder" makes the categorical statement that mental disorder is a disease of old age.⁴ From a study of the Massachusetts statistics he has demonstrated

that the age curve at incidence of admissions to mental hospitals closely parallels the death curve. This is again an illustration of the fact that just as the physical organism loses its elasticity so does the personality lose its ability to adjust, and show increasing tendency to undergo fracture. The reasons for this increase are not, however, entirely due to the fact that the number of persons in the upper age groups has shown an increase. It is, of course, true that such is the case. In 1900, for example, 4 per cent of the population were over 65 years of age, whereas by 1935 the proportion had risen to 6 per cent, and it is estimated that by 1980 approximately 15 per cent may be expected to fall in this age group. However, the reasons for this changing distribution are not to be found in the improved health of old persons. They are, rather, due to the decrease in infant mortality and in the incidence of epidemics, to the improved methods of treatment, to the decrease in accidents, and to the decrease in immigration. There is very little evidence that degenerative diseases in the upper age brackets are showing any tendency to diminish.

However, a large factor in the increased admissions in the older age group is probably to be found in the changing living conditions of the population. More and more there is a tendency to urbanization, to the giving up of homesteads and of single houses in favor of apartments or hotel life. Under these circumstances with increasingly close contact with one's neighbors, aberrant conduct, a tendency to wander, to be noisy, or to accost strangers, cannot be so well tolerated. Another factor which cannot be entirely overlooked is the decreasing respect for authority and for age being shown in the present generation and a tendency to an increasing looseness of family ties. One constructive factor in the present scene however, is the growing provision for pension systems for older persons. The development of these pensions should do much toward reducing the dread of dependency which is a serious psychologic hazard to the aging, and should likewise exert an effect in prolonging the time during which the borderline case of senile mental disorder may be cared for outside a mental hospital.

INVOLUTIONAL MELANCHOLIA

Turning now to a consideration of some of the types of mental disorder met with in later life, and following a rough chronology, the entity known as involutional melancholia is one of the earliest to appear in those well past the meridian. This is characterized by rather profound depression occurring in the involutional period of life, that is in the case of women between the ages of approximately forty five and fifty five, and in men slightly later. This disorder was shown by the late Dr. H. D. Palmer, of Philadelphia to be associated with a clear-cut type of personality make up as revealed by the life history of the individual. Palmer speaks of the make up as rigid, characterized by such features as introversion, an obsessional character, marked repressions, sexual maladjustment, sadomasochistic traits and hyper-religious trends. We are dealing here, then, with a specific type of personality which under the stress of the physical readjustment involved in the changing function of the gonads and perhaps accompanied by arteriosclerotic changes and by various other psychic factors and somatopsychic causes which have been already suggested, undergoes a failure along well recognized lines. It is, in other words, not the physical changes to which the psychosis can be

attributed—they are merely a factor and perhaps not the most important factor in the debacle of a preexisting warped personality

In involutional melancholia the prodromal period is likely to be lengthy, indeed in nearly half of the cases studied by Palmer it ran from one to three years. During this prodromal period will be found irritability, peevishness, pessimism, a tendency to insomnia, depression, and tearfulness. Very frequently distressing sensations in the head and the gastrointestinal tract are complained of. The appetite is poor, and a progressive loss of weight may be noted. Gradually the symptoms increase in severity, feelings of deep unworthiness and of sinfulness are expressed, together with marked agitation, during which the patient wrings his hands, picks at his face, tears at his



Fig. 51 Involutional melancholia. Patient fifty four years old

clothing, and cries out in anguish. There are intense feelings of guilt, particularly the delusion of the unpardonable sin, there is often the belief that the intestinal tract has been "burned out" and that for this reason all food must be foregone. Not infrequently there are also ideas of persecution directed against the family or others in the immediate environment. In spite of the marked depression and the presence of these very distressing delusions, the patient remains in fairly clear contact with his environment, at least when his attention is gained. One of the gravest dangers of this disorder is that of suicide. When clouding of consciousness is present it is usually to be attributed to arteriosclerotic changes. The prognosis in involutional melancholia, formerly considered poor, has been substantially improved by electroshock therapy. Cases in which paranoid symptoms predominate do not usually

respond to this treatment, however. The course may be prolonged, sometimes running several years. In such cases it is obvious that home care is probably not to be recommended. If it is insisted upon, proper feeding and protection against suicide are the most important factors.

In the early stages the use of testosterone propionate in the male has been found to be of occasional help—the usual dose, as high as 10 mg in oil, is administered intramuscularly two to four times weekly. Prolonged narcosis by the use of sodium amytal intravenously has been found to give occasional good results. It is likely, however, that provided the physical condition of the patient warrants, the full blown case will benefit as much from electroshock therapy as any form of treatment. By the use of a relatively simple portable apparatus a current of low intensity may be passed through the temples, causing a convulsive state with complete loss of memory for the event. Treatments may be given at intervals of two or three days and not infrequently substantial improvement is noted after five or six of them. Coronary disease and hypertension are usually contraindications, and during the treatment efforts must be made to prevent fractures and dislocations. Kino and Thorpe, of England, have recently reported a rate of 73 per cent recoveries in cases of depressions of the involutional and presenile periods.¹¹ The administration of the treatment is a highly specialized procedure, and should not be attempted by the inexperienced or outside of a hospital.

NORMAL MENTAL CHANGES

It is generally recognized that certain mental changes may be looked for as accompaniments of the *normal* senium. These changes may occur at varying ages in different individuals, but are unusual before the age of about sixty five, instances are recorded in which they were astonishingly lacking in persons of ninety years. In general the first thing noticed is the slight decrease in alertness, a tendency to slow up, and a narrowing of the span of interest. Accompanying this, and at the start rather insidious, is the loss of memory, particularly in the field of spontaneous recall. The patient becomes somewhat absentminded, he forgets where he has put things, cannot remember names, and perhaps later on even forgets faces. This impairment of memory is most striking for recent events, the events in one's earlier life standing out in bold relief and often being described with considerable prolixity. A tendency to over talkativeness and to the relating of rather trivial tales of the "olden times" may be noticed together with a tendency to circumstantiality, and at times irrelevancy. A disturbance of the sleep rhythm is not at all infrequent, and the patient though somewhat drowsy, particularly after meals during the daytime, may find considerable difficulty in sleeping at night and may indeed show some little nocturnal restlessness, prowling about the house. A tendency to hoarding, with resulting untidiness of the room and of the person, may be rather conspicuous. There may be periods of confusion, during which the patient is out of touch with his environment. Ideas of persecution, mild or marked, may be developed, particularly with reference to members of the family. Sometimes, unfortunately, these ideas have a substantial basis in fact, but often they are groundless and represent a projection of the patient's loss of status as the directing force in the household. The tendency to night prowling may, particularly if accom-

panied by mild confusion, result even in going out of doors and becoming lost or the victim of an accident, or instead in an attempt to light a fire for the purpose of relieving the suffering of the patient as death from exposure. The same is true of the aged women who are men-

sion, as it usually does, a feeling of inadequacy to the situation. Although the sexual activity is usually reduced, sexual interest is

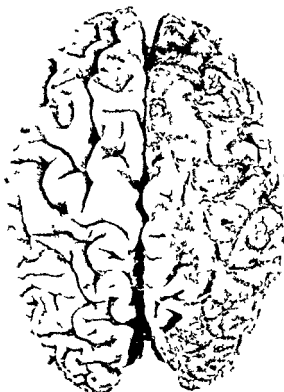


Fig. 52 Dorsal view of senile brain. The leptomeninges over the right hemisphere are somewhat thickened and fibrosed. Over the left hemisphere they have been stripped to show the shrinkage of convolutions and the widening of the sulci.

not uncommonly magnified, particularly in the male, as a compensatory mechanism. Fantasies are indulged in and sometimes translated into action in the form of fondling small children. In such cases legal action is likely, and sometimes takes the form of criminal prosecution.

The nocturnal deliria which are sometimes found in the aged may be accompanied by noisiness and may cause considerable annoyance to the neighbors. In such instances care at home becomes extremely difficult, if not impossible. Although delirium may be met in youth, maturity, or old age, it is a symptom which may develop on slight provocation in the aged, because of the relative instability of their homeostatic mechanisms. Anxiety

and fear are prominent together with loss of orientation and contact with the environment and the development of illusions and even hallucinations. On account of the patient's insecurity and anxiety Doty emphasizes the importance of reassurance and the avoidance of a confusing environment and of physical restraint. The nursing care is highly important; fractures must be avoided and nutrition and fluids must be maintained by nasal tube if necessary.

With the failing of mental acuity the judgment becomes impaired and the patient is rendered unduly susceptible not only to the wiles of scheming women but to business sharpers. In cases of this sort marriages of doubtful legality may be undertaken and wills often the subjects of later litigation may be executed (See Chapter 11.) The progress of the dotage as these symptoms are generally termed may be spotty. At times striking improvement

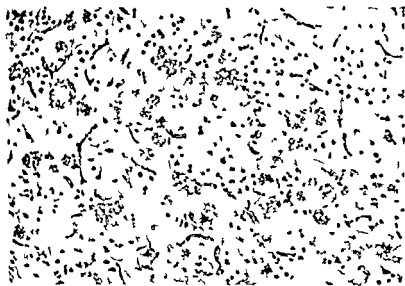


Fig. 53. Section of cortex from the same brain as shown in Fig. 52. There are many argentophilic plaques scattered throughout the gray matter (Braunmühl's silver nitrate technique).

may be noted for at least a short period, but this usually only in the earlier stages. On the other hand, an operation, accident, or an intercurrent illness may cause a rapid acceleration of the symptoms. The importance of not exposing the aging person to undue physical hardships or risks cannot be

sclerosis seem to respond with remarkable speed to the administration of vitamins, particularly niacin and ascorbic acid. These facts indicate that the vitamin reserve of aging persons is lowered, even to the danger point more than is the case in the average American adult. As a result a limitation in diet such as is not infrequently met with in senile persons by reason either

of inadequate dentures or failing appetite may result in an acute but sometimes striking psychotic episode. The prophylactic value of a fortified diet, with particular reference to fruit juices and brewer's yeast, should be borne constantly in mind.

Aging persons also seem to be more susceptible to acute drug intoxications than persons with a more active eliminative mechanism. For this reason sedative drugs in general should be used as sparingly as the conditions of the case permit. Acute deliria from the use of bromides, barbiturates, paraldehyde, or other sedative drugs, are probably more common than is generally realized, the symptoms being attributed merely to the aging process rather than, as may be the case, to the cumulatively toxic effect of the drug. For practical purposes, if sedatives are called for, sodium amytal ($1\frac{1}{2}$ grains) is probably the preferable drug (Chapter 7). Elixir of luminal (2 drams) or paraldehyde (10 cc) may also be considered, the latter being especially recommended by Doty.

Most of the symptoms which have been hitherto described can be cared for outside of a mental hospital. They cannot always be cared for, however, in the home. For one thing, the presence of small children may render it undesirable to have a dilapidated and untidy grandparent about the house, it may be, again, that both members of the next younger generation are at work during the day so that there is no one to care for the patient, again, there may be conditions such as noisiness, or a tendency to roam away, or sexual familiarities with small children, which render it impractical to attempt to keep the patient at home. A fair number of elderly persons who cannot get along at home can be cared for in the company of other old persons in homes for the aged or in boarding homes specializing in older persons.

OTHER MENTAL DISORDERS

Neuroses There are various conditions which may generally be denominated as neurotic which may be exhibited by the aging person. In these cases the symptoms of early senile deterioration which have been mentioned are in the background or perhaps absent. With the narrowing of interests and increasing idleness, there is a tendency to focus attention upon the various bodily functions, the excretory organs, the state of the complexion, the condition of the digestion, the regularity of one's heart action, and so on, may become a major preoccupation. Accompanying this may be a certain amount of depression and anxiety over the failing functions of the physical organism, and indeed suicide is not entirely unknown in this group. Again we may find acute anxiety states, periods of irritability, and what in a child at least would be called temper tantrums, manifested when the patient finds himself frustrated even in a minor degree by the activities of those about him.

In dealing with this group of disorders, the general principles of mental hygiene which have been laid down earlier in the chapter may be of assistance. Largely, the aim is to substitute interests and improve the situation which is evoking the condition, although psychotherapy should not be neglected. Proper attention to any physical complaints is important, dealing with the environmental situation is equally important—it may perhaps be the one thing which is needed to solve the entire problem. It must be borne in mind, of course, that the adjustability of the patient is not what it was forty years before, and cure even of these neurotic manifestations is sometimes very far

from complete. Nevertheless, much can be done if only the will to try exists on the part of the family and the physician. The borderline between the conditions already described and the frank and full blown senile psychosis is a vague one and it is rather difficult at times to say just when an aging person becomes definitely psychotic.

Senile Psychoses Several types of senile psychosis are enumerated in the textbooks and will be briefly described here merely for the sake of completeness. It should be borne in mind that here again the types are not always clearly separated, and that at different stages of the case a particular patient may appear to fall first in one category and then in another.

Simple deterioration has been already discussed under *dotage*. It exhibits merely a somewhat aggravated type of the symptomatology seen in that condition. The progress may be rapid or slow, contact with the environment

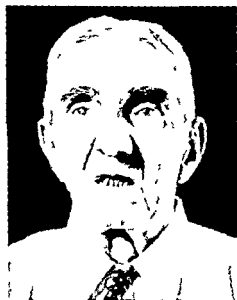


Fig. 54 Senile psychosis (simple deterioration) Patient eighty three years old

becomes less and less close, and finally the patient may become almost completely vegetative and totally out of contact with the environment, with untidiness, and progressive weakness, and death.

Another and less common form is the so called *delirious and confused type*. This is sometimes rapid in onset. There are marked restlessness and resistiveness, consciousness is much clouded, hallucinations are frequent, and exhaustion may supervene rapidly followed by death. A case of this sort is obviously one for a hospital where the best of nursing care can be provided. In some instances a striking response is noted to the administration of vitamin therapy.

In the *depressed and agitated type* we find, as the name implies, the presence of agitation together with delusions of sinfulness and nihilism and a depressive emotional tone. Here again on account of the agitation and resulting exhaustion, the course is likely to be short, and hospital care is

indicated, not only on account of the agitation, but on account of the possibility of suicide

The *presbyophrenic* type of senile psychosis is characterized by very marked loss of memory for recent events, together with filling in of the memory gaps by fabrication. It is more frequent among women than men, and is likely to occur particularly in those persons who were formerly marked by energy, vivacity, and good adjustability. The patient is likely to be friendly although occasionally irritable for short spaces. She is talkative and apparently alert, but when it comes to conversation it is found that she is almost completely out of touch with her environment. She is highly suggestible, and the most fantastic tales of exploits may be developed by suggestion, likewise, there is a considerable amount of purposeless activity. Recognition of the defects of memory is almost completely lacking in these cases.

The *paranoid* type, as its name implies, is characterized particularly by delusions of persecution. Again, we find here a marked exaggeration of traits which have been noted in the individual in his prepsychotic state, that is, the paranoid type of psychosis is likely to occur in persons who have always manifested a certain amount of dissatisfaction and other defense mechanisms, blaming their difficulties upon others rather than upon themselves. As the degenerative changes take place in the organism, the defenses which have been erected need strengthening, and as a result we find the exaggeration which characterizes the psychosis. Memory loss in this type is not so conspicuous as in the other types of mental disorder, although careful probing will usually indicate its presence. As the memory defect develops, however, the delusions may become more fixed and more absurd in a further effort to compensate for the failing powers of the organisms. Many of the ideas expressed by patients in this group involve members of their families, they are convinced that they are being robbed and otherwise discriminated against. Sometimes an attempt may be made to carry the delusions into action, as in the form of homicidal attempts, but these are comparatively rare. In some instances a person of this sort can adjust himself to a group of persons of his own age away from his family, but occasionally, particularly if threats of violence are made, commitment to a mental hospital becomes necessary.

The form sometimes classified with the senile psychoses known as *Alzheimer's disease* or *presenile psychosis* may be mentioned here in spite of its relative rarity. There is a rapid mental deterioration, with restlessness, delirium, confusion, and rather rapid progress, with onset as early sometimes as the fifth decade. The neurologic findings, particularly at autopsy, are definite, and the age of onset may be much earlier than the senile period.

Another rare form of presenile psychosis is known as *Pick's disease*. Here again there are marked changes in the structure of the brain, the symptoms are rapid in development and dementia develops early in the course of the disease. In spite of the striking dementia, memory may be well preserved, at least in the earlier stages. Focal symptoms are sometimes noted, such as *agraphia* and *aphasia*. The condition goes on to early inability to leave the bed, incontinence develops, dementia becomes extreme, and the patient usually dies within a few years of some intercurrent infection. Obviously both of these types are hospital problems. (See Chapter 18.)

In general it should be borne in mind that in the senile group hospital

care should be considered only after all other possibilities have been exhausted. The sudden change of environment and of manner of living involved in commitment to a mental hospital often is hard for the patient, and it is for this reason that caution is advised. In general it may be said that the patient who shows antisocial conduct in the form of homicidal threats or in the form of sexual advances to small children should be committed. Another indication is a persistent tendency to roam away so that there is substantial danger of accident. A third indication is the condition in which the patient is such a serious nursing problem because of untidiness, restlessness, and noisiness that he cannot be cared for at home within the means of the family.

Arteriosclerotic Psychosis In the group just discussed we are dealing partly with diffuse changes in the brain, partly with psychologic mechanisms, and partly with the general aging of the physical organism. There are certain conditions in which arteriosclerosis of the brain, either focal or diffuse, causes mental symptoms which should be considered here. The distinction between the arteriosclerotic and the senile psychoses is to a large extent academic, in either case we are dealing with the process of aging. Very few patients with senile psychosis have wholly escaped the development of arteriosclerosis. In general, however, for purposes of classification, cases in which cerebral arteriosclerosis is readily demonstrable are classified as psychosis with cerebral arteriosclerosis rather than as senile psychosis.

The case is one in which the disorder is not a function of the type of cortical damage but is conditioned very largely by the preexisting personality makeup. In the earlier stages of the disorder a striking fluctuation in symptomatology may be noted almost from day to day and at times a tendency to depression may be noted. Poor judgment in business affairs may be a practical result of the failing attention. At the same time there may be complaints of headaches or of dizziness, not infrequently the physician, particularly if an ophthalmoscopic examination is not made, may pass these off as neurasthenic in character and advise a change of scenery ("touristic therapy"). Alvarez attributes many of these complaints in the elderly to "small strokes," that is, minute cerebral hemorrhages which may elude recognition because they do not cause such classical signs as hemiplegia or speech defect. The patient may suffer nausea, headache, or dizziness, and concomitantly display apathy, carelessness, and other persistent changes in personality. Changes in the ethical attitude may be noted and there may be an increase in alcoholic indulgence or in other flagrant and public types of misbehavior. Childishness and obstinacy may be noted, and memory disturbances are not infrequent. Attacks of irritability become common, and at times there is found a tendency to easy and unprovoked weeping. The distressing feature of these symptoms is that the patient may recognize them as pathologic, a fact which emphasizes his depression and his worry concerning himself and his future. The development of delusions is not at all uncommon, sometimes hypochondriacal and sometimes persecutory, ideas of jealousy are not infrequent. Rothschild has demonstrated effectively that the type of symptomatology noted is not wholly a function of the type of cortical damage but is conditioned very largely by the preexisting personality makeup. In the earlier stages of the disorder a striking fluctuation in symptomatology may be noted almost from day to day.

In the type of cerebral arteriosclerosis in which the larger vessels, particu-

larly those at the base of the brain, are involved, one of the very early symptoms may be a cerebral hemorrhage, an attack of aphasia, or some other focal disturbance, such as the fleeting loss of power in an extremity. Headaches, particularly in the region of the vertex and more severe in the morning, fatigability, and intolerance of alcohol become striking, attacks of vertigo and of emotional lability, particularly in the form of weeping at trifles, are noted. Station may become more insecure, the gait uncertain, and the steps short and spastic. Tremors may be present, and pupillary inequality is not uncommon. Apoplecticiform or epileptiform attacks may occur. In cases of advanced arterial hardening, a sudden fall in arterial tension may bring about symptoms much like those due to vascular spasm. Such hypotension may be due to narcosis, excessive vasodilator medication, or sudden cardiac decompensation, for example. (See Chapter 30.)

The immediate and subsequent care of cases of hemorrhage or cerebral embolism has been discussed elsewhere in this volume. In most of the cases a certain amount of residual paralysis, sometimes total, remains, and there is also the possibility of aphasia, *agraphia*, or *alexia*. Furthermore, a certain amount of loss of mental acuity usually persists following a cerebral insult, and sometimes this is so marked as to be properly termed a dementia. There may be difficulty in carrying the train of thought, with silliness, and only a very superficial contact with the environment. It is extremely important to avoid overinvalidization following a cerebral insult, since there is a tendency to hypochondriasis and self pity which must be combated. The patient should be encouraged to do as much as his physical condition will permit. In a resultant aphasia or *alexia* steps should be taken provided the patient is at all cooperative, to reeducate him. This is a lengthy process, and constant encouragement will be required. Much, however, can be done by persistent efforts along these lines. In dealing with the irritability and the fretfulness which sometimes are found in the hemiplegic, attention to the mental hygiene of the rest of the family is to be recommended.

From the preceding discussion of the senile, involutional, and arteriosclerotic mental disorders, the impression should not be gained that the senium is a guarantee against any other type of mental disorder. Such, of course, is far from the case. Episodes of manic depressive psychosis are not at all unknown during the senium, although the history will almost always reveal previous attacks earlier in life. Again, general paresis may develop late in life, in one study 10 per cent of the first admissions were found to be in those over sixty years of age.¹ Some of the signs of this disorder, such as the Argyll-Robertson pupil, the tremulousness about the mouth and tongue, and the characteristic speech defect, are to be distinguished from the types of neurologic signs often found in the arteriosclerotic. Sometimes the symptoms of other types of psychoses occurring during the senium are modified by the senile changes, particularly the loss of recent memory. This is especially noted in hospital patients who have undergone the senium while in hospital residence. For the purposes of the private practitioner, however, cases of this type are of negligible importance.

PROGNOSIS

In general the prognosis for any mental disorder occurring in the later reaches of life is not particularly good. This applies especially to the senile

and arteriosclerotic group, where we are dealing with physical changes in the central nervous system which are irreversible in character. Much can be done, however, to make the patient much more comfortable both to himself and to those about him. Particularly should the mental hygiene aspect of the aging process be borne in mind, as already outlined. Emphasis should be placed upon the compensating factors in aging—the balanced judgment based on experience, the opportunity for reflection and study, and the development of hobbies afforded by leisure (See Chapter 5). With the inculcation of a proper attitude on the part of the patient, with the avoidance of overinvalidization, and by the exercise of patience, tact, and sympathy, the physician and the family can do much to make the transition from late maturity to the senium considerably easier both for the patient and for his family.

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CHAPTER 18

ORGANIC DISEASES OF THE BRAIN, SPINAL CORD AND PERIPHERAL NERVES

CARL D. CAMP

IN the nervous system, involution and the so-called diseases of involution begin at about the age of sixty-five to seventy. In some cases they may begin as early as the age of twenty but it is well known that centenarians may show little evidence of degenerative change. We cannot say whether this variation is due to disease, to something in the previous mode of life, such as various infections or intoxications, or to an increase in the vulnerability of the nervous system to such conditions. Perhaps we should look upon these degenerative diseases as *abiotrophies*, a term used by Sir William Gowers to explain some of the familial degenerative diseases such as Friedreich's ataxia. According to this view, the inherent vitality of the cell itself is the determining factor in its life span and consequently the important factor in the degeneration of the tissue. The factor of nutrition is also important in this connection. Whether this is a result of digestive disturbances and of malnutrition of the body as a whole or of changes in the metabolism of the individual cell is not determined.

I shall not attempt to describe the changes in the nervous system in "normal" old age, if there is such a thing. The changes that have been described as normal could just as well be regarded as pathologic. The thickened meninges, cortical atrophies, widened ventricles, pigmentary changes in the nerve cells, corpora amylacea, and so on, can be looked upon either as pathologic or as simply changes natural to old age.

When we try to classify certain diseases of the nervous system as senile we meet even greater difficulties. In fact, in the present state of our knowledge it is impossible to say what is a "disease of old age" and what is "disease occurring in the aged." It is also important to realize that many conditions common to earlier years such as injuries or brain tumor may occur also in the aged and that the symptoms and signs may be modified by the age changes, not only in the nervous system, but in the bones, joints, muscles, and vascular system.

In this chapter I shall discuss those conditions which, on the basis of clinical experience, seem to be important to those interested in geriatrics from a practical point of view.

CEREBRAL ANEMIA AND HYPFREMIA

Cerebral Anemia. The brain acts best only when it is supplied with a

• • • • • There are

arteries, so that the stoppage of any one will be compensated through the

circle of Willis. It is, therefore, rare to have a cerebral anemia due to an acute stoppage of the blood supply. However, anemia of the brain may occur either acutely and temporarily, or chronically as the result of various other conditions. The principal causes are a deficient supply of blood to the brain, due to heart failure, a low blood pressure, or a sudden dilation of the abdominal blood vessels so that the blood is transferred to the so called "splanchnic pool." A poor quality of the blood, especially when deficient in oxygen or sugar content, may cause much the same symptoms as a cerebral anemia. Permanent cell changes in the brain depend upon the duration and severity of the condition. According to Gildea and Cobb,¹ fifteen minutes of severe cerebral anemia will cause permanent cellular changes.

The principal symptoms of cerebral anemia are vertigo, tinnitus, clouding of consciousness followed by coma, cold and clammy skin, nausea and vomiting, and occasionally convulsions. In old age the principal cause of cerebral anemia is a failing heart, but the conditions that may cause it in the young occur almost as frequently in elderly persons, i. e., loss of blood, anoxia, hypoglycemia, serious injuries especially to the abdomen, and so on. Cerebral arteriosclerosis may also be a cause of cerebral anemia and this condition of course is much more common after the age of sixty five.

If the anemia is temporary the prognosis for recovery is good but measures must be taken to prevent recurrences.

The treatment is directed to the removal of the cause, but may include the use of stimulants, lowering the head, and blood transfusions if necessary.

Cerebral Hyperemia. Active cerebral hyperemia occurs in some cases and the symptoms are similar to those of cerebral anemia. At one time it was thought that cardiac overaction, sunstroke, the inhalation of some gases and similar incidents could induce an active congestion of the brain. This is doubted by some observers.

Passive congestion of the brain due to some obstruction to the venous outflow is likely to cause drowsiness, vertigo, headache, mental confusion, excitement or depression and occasionally convulsive seizures. Any condition interfering with the venous return of the blood from the head may be a cause: chronic cardiac and pulmonary disease, pressure from glands or tumors in the neck, the wearing of a tight collar, and similar situations. In elderly patients even a recumbent position may cause cerebral symptoms, as in the case cited by S. A. K. Wilson of an eighty-four-year old man who had to sleep sitting up because of "startling visual hallucinations" as soon as he would lie down. I have recently observed a similar case. The phenomena caused by cerebral hyperemia are usually transient but they may persist until the cause is removed.

CEREBRAL ARTERIOSCLEROSIS

The familiar aphorism that a man is as old as his arteries has been thus modified by Rolleston:² "the condition of his arteries provides an index not so much of his age as of his adventures."

It must be recognized that there are different kinds of sclerosis of the arteries, some of which may be found at an early age. In the so-called senile or involuntal type of arteriosclerosis the muscular coat of the medium size arteries atrophies and is replaced by connective tissue. At the same time the cells of the intima increase, they may degenerate and lime salts be deposited

in the arterial wall. These changes may occur in varying degrees in different arteries. I have seen cases at autopsy in which the cerebral arteries are markedly sclerosed when the arteries in the rest of the body remained in a fairly normal condition. The opposite relation can also be found.

Etiology. In the genesis of arteriosclerosis toxic or toxic infectious factors certainly play a prominent role in many cases. I am inclined to believe that the removal of local infections would be important in preventing or delaying such conditions. Oppenheim³ claims that persistent vasomotor hypertonia of affective origin is an important cause of cerebral arteriosclerosis. This I believe to be a matter of common observation. It is not always recognized that a life full of worry, nervous strain, and intense cerebral activity will lead to cerebral arteriosclerosis. Overeating and the use of alcohol have been blamed for arteriosclerosis and may be a factor in some cases, but they are certainly not as important as formerly thought.

Symptoms. The relation of the symptoms in cases of arteriosclerosis to the findings at autopsy is often difficult to explain. Severe changes in the arteries may be found at autopsy in cases where apparently the patient has had no symptoms of the condition, or at least none that have been recognized by his associates or himself.

Early symptoms may include headache, often transient, dizziness, fatigue, somnolence, failure in memory especially for certain things, general irritability and mental depression with emotional lability. In many cases the condition can only be diagnosed with certainty when the patient begins to have focal symptoms either from cerebral thrombosis or cerebral hemorrhage.

In advanced cases, mental deterioration may become much more marked. The patient may show changes in character and conduct, delusions of nonsystematized but persecutory type, and finally a complete dementia. These patients even in the last stages, may be found to be intelligent in some respects or show flashes of intelligence on some occasions. This may help to differentiate the condition from senile dementia. A normal or even a low blood pressure does not rule out the diagnosis of cerebral arteriosclerosis.

Prognosis. The prognosis of cerebral arteriosclerosis is bad for recovery. Since the symptoms, at least in their earlier stages, are often due to some factor which is added to the arteriosclerosis it is quite possible that the patient may symptomatically improve although the condition of the artery remains unchanged. Such added factors might be nutritional disturbances, lack of vitamins, or to the use of some sedative medication which was originally given for the relief of symptoms such as irritability or insomnia.

Treatment. It is desirable to relieve the patient of all mental stress and strain and to substitute more simple routine duties for his previous occupation. All sources of toxemia or infection should be eliminated. This investigation should include among others the tonsils, sinuses, gallbladder, appendix, prostate, and Meckel's diverticulum. The bowels should be regulated by small doses of calomel if necessary, or by small doses of cascara sagrada or some similar laxative, mineral oil is not advisable. Vitamins, particularly of the B group, might be valuable, especially to stimulate the appetite. Should the patient have become adjusted to a high blood pressure it is not desirable to use active measures to lower the blood pressure considerably. A high blood pressure is often a provision of nature to force blood through the narrowed arteries so that if the blood pressure is radically lowered a cerebral

thrombosis may be the result. The use of sodium or potassium iodide, either by mouth or intravenously, may be of some benefit (See Chapter 30)

ACUTE CEREBRAL VASCULAR LESIONS

The term 'stroke' is commonly applied to three forms of acute cerebral vascular lesions: cerebral hemorrhage from a bursting blood vessel, occlusion of a vessel by an embolus, and a thrombosis of a cerebral vessel which either stops the flow of blood completely or lessens the flow through that artery to a point where the blood supply to the part is not sufficient to enable it to continue to function. Since the mechanisms producing these three conditions are entirely different and the treatment equally so, it is important to

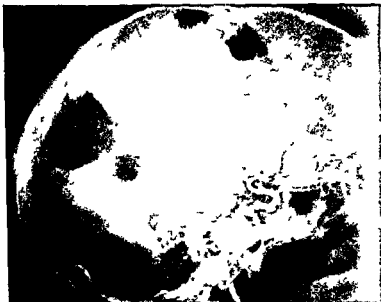


Fig. 55 Arteriogram showing thrombosis of the posterior branch of the mid-cerebral artery. The patient, a seventy-year-old woman, was struck on the head and developed a hemiplegia about twelve hours later. This history suggested a diagnosis of subdural hematoma. No hematoma was found and an arteriogram showed the correct diagnosis. (From the Department of Neurosurgery, University of Michigan. Courtesy of Dr. Robert Bassett.)

distinguish between them. Cerebral thrombosis is the only one that is really common in old age.

Cerebral Hemorrhage The essential cause of intracerebral hemorrhage is the concurrence of a weakened blood vessel wall with an increased blood pressure. If the vessel wall is entirely sound, a cerebral hemorrhage would result only from the most extreme hypertension, but if the vessel wall is weakened, even a normal blood pressure may cause a rupture, especially if the blood pressure is raised suddenly. Such conditions are much more common between the ages of forty-five and sixty and are rare in older people. In the history, it is usually noted that the attack comes on while the patient is active or under some sudden strain. The shock effect of a cerebral hemorrhage causes a sudden loss of consciousness as if the patient had been struck

a blow Frequently this is accompanied by other evidences of shock The resulting unconsciousness may be short or long depending upon the size of the hemorrhage Usually the result is a hemiplegia due to the destruction of the internal capsule and of the pyramidal tract at that point Most common site for hemorrhage is the lenticulostriate or lenticulo optic artery, and these arteries have been called "the arteries of cerebral hemorrhage"

Cerebral Embolism An embolus may occlude a cerebral artery at any age the most common cause perhaps is vegetative endocarditis or pulmonary disease Other sources of embolism such as infection, air embolism, etc., may occur The symptoms are abrupt and much like those of the cerebral hemorrhage The condition is comparatively rare after sixty It may occur in cases of auricular fibrillation but in most of the cases of that sort that I have seen the correct diagnosis was cerebral thrombosis due to the sudden lowering of the blood pressure

Cerebral Thrombosis In approximately 90 per cent of the so-called apoplectic strokes occurring in individuals more than sixty-five years of age the cause is cerebral thrombosis In such cases the arteriosclerotic change, if accompanied by intimal proliferation, reduces the lumen of the vessel, especially the smaller branches, and under such circumstances, the flow of blood through the arteries may reach a point at which the volume is not sufficient to preserve the function of the parts The word thrombosis in such cases is somewhat of a misnomer although if the blood flow ceases entirely actual thrombosis will occur According to Gildea and Cobb "the merest trickle of blood will sustain nerve cells for a long period" However, the function of the cell may be destroyed long before an anatomic change is detectable In most cases the onset of the condition is brought about by reduction in blood pressure This may be sudden such as would occur in surgical shock or spinal anesthesia, or it may be gradual as the result of prolonged rest in bed The area of the brain affected may be large or small depending upon the size of the vessel Usually these are cortical vessels and the effect is limited to the area involved In some cases the motor area may be affected causing paralysis or the postcentral convolution causing a limited anesthesia, or the cuneus region causing hemianopsia, or the effect may be on some so called silent area of the brain and cause no apparent functional disability Because the area of destruction is likely to be much smaller than in cerebral hemorrhage and there is no special shock, it is not likely that there will be a loss of consciousness In most of these cases the patient awakens in the morning after a good night's sleep and finds himself partly paralyzed Premonitory symptoms may occur if the patient is up and about They consist of vertigo, mental confusion, transient aphasia, or numb sensations in the extremities If the blood flow is established within a short time complete restoration of function will occur

The *prognosis* is doubtful Immediate death from cerebral thrombosis is unlikely but the fact that such a condition is present indicates that probably other cerebral arteries are similarly affected One can, therefore, expect recurrences of the thromboses or involvement of additional arteries and areas of the brain

Treatment must be directed to increasing the lumen of the cerebral arteries if possible and also to efforts to raise the blood pressure or to keep it up to a point where the blood is forced through the narrowed arteries The

use of iodide has been advised in such cases but the method of its action has never been demonstrated. Clinically, however, the effect of large doses of iodide may be surprisingly favorable especially if it is given intravenously. For such use the dose is usually 30 grains of sodium iodide in 20 cc of water, repeated every four hours. Rest in bed is not indicated unless the condition of the lowered blood pressure could be attributed to a failing heart. Stimulants are useful especially undiluted whisky in good doses. Strychnine and caffeine may also be used. Moderate excitement does no harm to the patient as it would in a case of cerebral hemorrhage. Although in some of these cases, especially when there is a marked general arterio-sclerosis, the blood pressure may be high it is certainly better not to try to lower it.

The *aftertreatment* in these cases should include massage and passive movements of the paralyzed limbs and as much activity as the patient is able to take without fatigue.

Pseudobulbar Palsy The occurrence of multiple areas of cerebral softening affecting both sides of the brain may give rise to a so called pseudobulbar palsy. It should be emphasized that the word "pseudo" modifies bulbar and not palsy. The difficulty in talking and swallowing is certainly real. In contrast to true bulbar palsy this condition usually occurs after sixty, and the history of the case will include an attack of hemiplegia or partial hemiplegia which affects one side of the body and then the history of a second attack affecting the other side. The difficulty in speech and deglutition develops suddenly after the second attack. The condition may be differentiated from true progressive palsy by the absence of atrophy of the tongue and fibrillary tremors in these cases. Patients with pseudobulbar palsy also frequently show an instability of emotional expression such as forced laughing and crying due to the involvement of the basal ganglia.

The prognosis for life in pseudobulbar palsy is not bad except for the danger of additional areas of softening. The treatment is the same as that for cerebral thrombosis. The dysarthria and dysphagia will have a tendency to improve rather than get worse.

Cerebral Arterial Spasm Spasmodic contractions of cerebral arteries undoubtedly occur and have been observed during operations on the brain. By some they are regarded as the cause of migraine attacks and of epileptic fits. However, the assumption that arteriosclerotic vessels are particularly prone to spasm is certainly doubtful. The fact that their muscular coats have lost their contractibility by undergoing fibrous degeneration is certainly against the idea of increased tendency to contraction. Genuine angiospastic affections are more common in young or middle aged persons. Symptoms suggesting this condition occurring in the aged would suggest cerebral arteriosclerosis and forebode thrombosis.

Intracranial Aneurysms Intracranial aneurysms are not common in old age and when they do occur in the senile they are part of cerebral arterio-sclerosis. In a collection of thirty three cases reported by Fearnside⁴ only three appeared after the age of seventy. Cerebral aneurysms are either congenital or occur between the age of forty and fifty years. In a review of 1125 cases collected from the literature, McDonald and Korb⁵ found that the greatest incidence was between the ages of forty and sixty. The oldest patient, however, was eighty seven.

DEGENERATIVE DISEASES OF THE BRAIN

The so-called senile dementias are characterized by a diffuse atrophic change in the brain. The mental and physical symptoms which may lead to the diagnosis of this condition often are much more marked than the gross pathologic changes would explain. It may therefore be assumed that other factors must play an important part in the cause of the disabilities of the

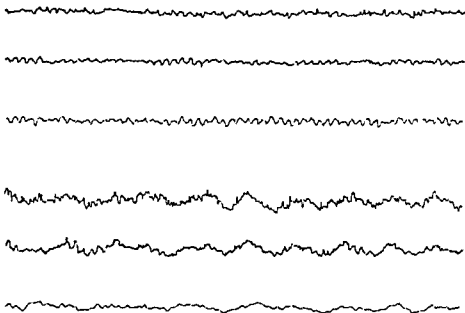
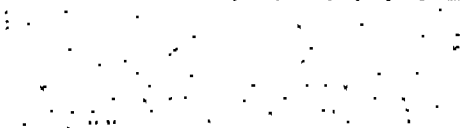


Fig. 56 Electroencephalographic tracings. The three upper tracings are practically normal with leads from the inferior frontal region to anterior temporal, frontal to posterior temporal and frontal to occipital. The three lower tracings are from the same locations of leads in a case of Pick's disease. The electroencephalogram may be of some value in the differential diagnosis between Alzheimer's disease and Pick's disease. In Alzheimer's disease the abnormalities are minimal and are not localized. In a case of Pick's disease the electroencephalogram was reported as follows: Special localization type of EEG done



individual (see p. 265). When such factors as malnutrition, perhaps vitamin and sugar deficiencies especially, various toxic states, the condition of the circulation and especially emotional factors are adequately investigated and corrected, the patient's condition may be considerably improved. It must be than curative and that general the degenerative it have special features

Pick's Disease. This is a special form of cerebral atrophy named after Arnold Pick, who attempted to prove that senile brain atrophy might be

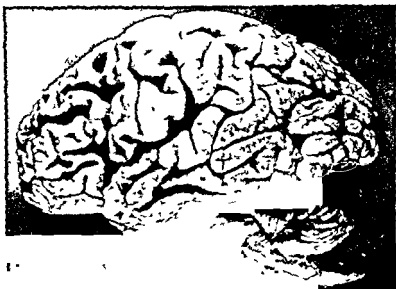


Fig. 57 Photograph of the brain from a case of Pick's disease. Note the frontal and temporal lobe atrophy with relatively normal appearance of the rest of the brain (From the Neuropsychiatric Institute, University of Michigan. Courtesy Dr. K. Scharenberg.)

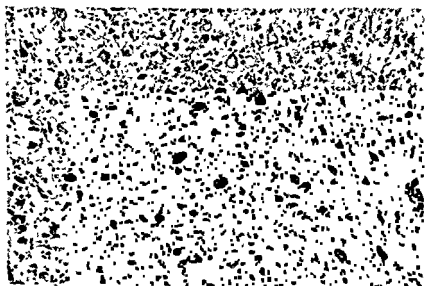


Fig. 58 Photomicrograph of a section from the cortex in a case of Pick's disease. Nissl stain. Showing inflated nerve cells (From the Neuropsychiatric Institute, University of Michigan. Courtesy of Dr. K. Scharenberg.)

accompanied by marked focal symptoms. The lobar atrophy of the brain, chiefly frontal and temporal, found at autopsy in these cases sets this condi-

tion apart as a somewhat distinct clinical and pathologic entity. Apparently it is more common in women than men and it may begin as early as the age of forty. However, one case is reported to have begun at the age of ninety-one.

The symptoms vary with the part of the brain affected. The most striking and frequent symptom is memory defect, which comes on gradually and is often first noticed by the mistakes that the patient makes in his work. The patient is inclined to be apathetic and indifferent. Focal signs such as hemiplegia, aphasia, apraxia, and so on come gradually. It is often noticed that the apraxia is greater than agnosia. The condition is regarded as a parenchymatous involutional change which affects some parts of the brain and not others. Differential diagnosis from senile dementia is difficult except that in these cases focal symptoms are more prominent and the onset may be earlier in life. They often give the impression of being more insane than either the senile dement or the arteriosclerotic.



Fig. 59. Photograph of the brain from a case of Alzheimer's disease. Note the generalized atrophic change. (From the Neuropsychiatric Institute, University of Michigan. Courtesy Dr. K. Scharenberg.)

There is no treatment that is effective in controlling the course of the disease.

Alzheimer's Disease In 1917, Alzheimer published a paper entitled "On a Peculiar Disease of the Cerebral Cortex." In these cases the brain appears to be diffusely atrophic on gross examination but the microscopic changes are peculiar and characteristic. The so-called senile plaques have an

There is also
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monly between the ages of fifty and sixty but Malamud and Lowenberg reported a case which began when the patient was fifteen. Death ensued at twenty-four and the diagnosis was based on the finding of the characteristic plaques at necropsy.

The disease begins insidiously, usually between the ages of fifty and sixty, with a slight defect in memory and behavior. Ideas of space and time are equally likely to be faulty. Mistakes are made in dressing, eating, working, and in speech. Later in the disease the speech may become a jumble of words—the so-called vocal clonia. Agnosia and apraxia are about equal in degree. Many of these patients show increased activity. They are always busy but accomplish little. Occasionally they are hypomaniac. The condition lasts from five to ten years and the treatment is palliative or institutional.

Parenchymatous Atrophy of the Cerebellum. This rare disease begins at an average age of sixty-two, the limits being between forty and eighty years. It may begin suddenly. It is not usually hereditary although Schuster⁶ reported a family with five cases, all occurring in the sixth decade. The principal symptoms are the difficulty in standing and walking and the ataxia in the hands. Speech is also affected but the mental powers are usually preserved.

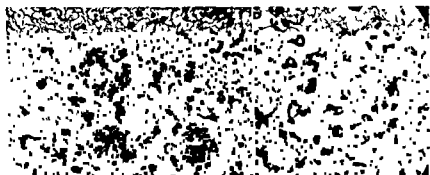


Fig 60

Fig 61

The patients may live for many years. The chief pathologic change is in the Purkinje cells of the cerebellum. The process is regarded as an involutional change and treatment is futile.

PARKINSON'S DISEASE

(Parkinson Syndrome, Shaking Palsy, Paralysis Agitans)

In 1817, James Parkinson described "shaking palsy," a condition characterized by a rhythmic tremor of the extremities and a gradually increasing stiffness of the entire musculature. Earlier reports confused this condition with multiple sclerosis, but it was clearly differentiated from this disease in 1892 by J. M. Charcot. Wilson⁷ says that "it is not a disease of senility," but Mendel⁸ gives the average age of onset in women as fifty-five and in men at fifty-seven.

The disease is more common in men than in women and has been reported in all races and social ranks. Many observers have noted that the victims of the disease have usually lived a hard working life with few vacations and have not been addicted to the use of alcohol or tobacco.

Pathology. Parkinson's disease consists of a slowly progressive degenerative process affecting the corpus striatum and adjacent areas of the brain. Since the symptoms are due to the part of the brain affected rather than to the nature of the process, other pathologic conditions, located in the same region, will cause similar symptoms. Thus we may have the Parkinson syndrome (sometimes called pseudo-parkinsonism) as a result of chronic encephalitis, syphilis, trauma to the brain, an arteriosclerotic area of softening, or a cerebral hemorrhage in this same area. Such cases, of course, may occur at any age, but the true disease is a special type and location of a senile process and, in my experience, practically never comes on before sixty years.

Clinical Signs. The rigidity of the muscles may come on so gradually that it is not noticed by the patient. It is often more pronounced on one side at first, later becoming generalized. It causes a masklike rigidity of the face with rare winking and a slow smile. The arms are held flexed at the elbow and do not swing in walking. The knees are held slightly bent and the gait is shuffling, with short steps. Occasionally the body center of gravity gets ahead

*To Winifred
from Mamma*

This a specimen of my handwriting

Fig. 62 Handwriting in paralysis agitans. Notice the small size of the letters and the continuous tremor. The alignment of the letters is good and there is no ataxia. The upper specimen shows the normal handwriting about one year before the onset of the disease.

of the feet so that the patient is forced to walk forward until he can bring up against something to stop himself (festination). A similar phenomenon may be seen if the patient attempts to walk backward (retropulsion), or sideways (lateropulsion). All movements of the body or extremities are slow and difficult because of the stiffness, but it has often been noticed that if the patient is frightened or angry he can move rapidly in spite of these.

The tremor is a rhythmic type of fairly large amplitude and about 100

are normal.

The neurologic examination shows no atrophy or true paralysis and the reflexes are usually normal. There is no Babinski reflex or other pyramidal tract signs. Objective sensory tests are negative except that vibratory sensibility may

be diminished. Subjectively these patients often complain of various paresthesias but they are like those often noticed in senility. Bladder and rectal functions are not disturbed.

The differential diagnosis among cases of parkinsonism due to different causes is given in Table 9. The differentiation from cerebral softening is made by the gradual onset and the slow spread of the symptoms and by the presence of the characteristic tremor.

In paresis the blood and spinal fluid will show positive serologic reactions and there are definite mental disturbances.

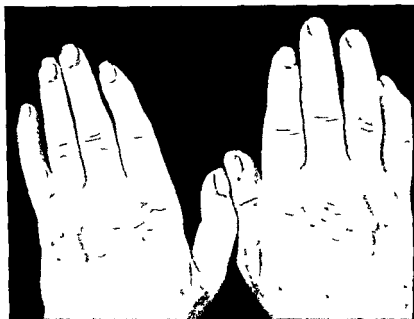


Fig. 63 The ulnar deviation of the fingers in a case of unilateral paralysis agitans. This was described by Dr. William G. Spiller as a characteristic of paralysis agitans and is useful in distinguishing it from postencephalitic parkinsonism.

Multiple sclerosis is definitely a disease of younger individuals; it is rare in those more than fifty. There is not the same muscle rigidity as in paralysis agitans and the tremor of multiple sclerosis is of the intention type.

Course and Prognosis. There is a gradual progress to an almost complete physical disability, but the disease is not fatal. There is no known cure but with careful management and judicious use of drug therapy the patient may continue to have a useful and comfortable life for many years.

Treatment. A most important point in the management of these cases is to keep the patient moving. As his muscles begin to stiffen and his movements slow, his natural tendency is to rest, but this in turn seems to induce changes in the muscle tissue itself.⁹ The patient must therefore be urged to continue his work and to take plenty of exercise. The mistake is often made of advising

these patients to retire and give up their work or business interests. Mental exercise seems quite as important as physical.

The drugs of the atropine series—hyoscine, scopolamine, atropine, and daturine—are of great value in lessening the tremor and rigidity. They have no curative value. Their effect varies greatly in different cases, so that it is my plan to try each one for a period of about two weeks in doses up to nearly the limit of tolerance.

After such a trial one can select the most effective drug for permanent use in that particular case. The patient himself will often show a most decided preference. It should be explained to the patient that taking the medicine is like wearing glasses. It may be a nuisance but it will not hurt him, and usually the dose must be adjusted from time to time to fit his condition.

TABLE 9

DIAGNOSIS OF THE PARKINSON SYNDROME ACCORDING TO ETIOLOGY

Paralysis agitans	Post encephalitic	Syphilitic	Traumatic	Vascular accident	Senile tremor or family tremor
Almost always more than 50 usually more than 60	Any age	Any age	Any age	At about 50	At about 50
No history of encephalitis	History of encephalitis but not always obtained	Positive serologic reaction or a history of syphilis	History of recent trauma to head	Hypertension	Usually a family history
Onset gradual	Onset gradual	Onset gradual	Onset rapid	Onset rapid	Onset gradual
Affects one extremity first. Then spreads. Usually does not affect head	Same as paralysis agitans	Same as paralysis agitans	Same as paralysis agitans	Same as paralysis agitans	Affects head first and then both hands
	Other post-encephalitic symptoms: oculogyric crises, ocular palsies, etc.	Other clinical signs of syphilis	Postconcussion syndrome: headache, dizziness, etc.	Often combined with partial and pseudo-bulbar palsy	Neurologic examination negative
Spinal fluid negative	Spinal fluid sugar increased in relation to blood sugar	Spinal fluid positive for syphilis	Spinal fluid negative or may contain blood	Same as the traumatic	Spinal fluid negative

Other similar drugs have had special advocates, the Bulgarian wine of belladonna, rabellon, genoscolamine, and others, have similar effects and may have a special value in certain cases, but in my own experience I have not found this so. Vitamins of the B group have been used in the treatment of this condition, but I have not found them to be of any value in genuine Parkinson's disease, although they may increase the patient's feelings of strength and well being. Benzedrine is another drug that seems to be of more value in post-encephalitic cases than in the degenerative type. Sedatives, especially the barbiturates, are of some value in the treatment of the tremor and weakness, but they are of little value in the rigidity. Whisky

Physiotherapy is not of much value. Massage, especially vibratory massage to the stiffened muscles, has only a temporary effect and baths also relax the patient for a few hours only.

Constipation is a common accompaniment of the condition and should be combated by diet, laxatives, preferably of the vegetable type such as aloes or cascara sagrada, and by enemas if necessary

OTHER DISEASES OF THE BRAIN OCCURRING IN OLD AGE

Brain Tumor. This is rare in persons more than sixty five. A survey of statistics furnished by Cushing, Downman and Smith and others shows less than 2 per cent. It is quite probable that the percentage would be higher, but for the fact that such cases occurring late in life may be misdiagnosed as senile degenerations or chronic vascular brain disease. Moersch, Craig and Kernohan,¹⁰ in reporting one hundred cases of tumor of the brain in patients more than sixty years of age, found that glioblastoma, meningioma, and acoustic neuroma accounted for 82 per cent of all the cases. They emphasize that the variability of the symptoms in these patients makes the diagnosis difficult. The most important diagnostic feature is the presence of choked disk, but this may be late in appearing or even absent in some cases of brain tumor. When choked disk is superimposed on visible arteriosclerotic changes in the fundi the differential diagnosis from hypertensive retinitis may be impossible.

The treatment of tumor of the brain is surgical. Experience has shown that elderly patients stand these operations amazingly well, especially when they can be done under local anesthesia and with modern technic.

Cerebral Syphilis. Syphilis of the brain is not uncommon in old age. It must be remembered that not infrequently syphilitic infections are acquired after the age of seventy. The treatment of the early stages at that age should be conservative. It is my opinion that cerebrospinal syphilis in advanced years should also be treated conservatively with iodide and mercury or penicillin rather than arsphenamine or malaria. I have seen several cases of late cerebrospinal syphilis, tabes, and taboparesis in elderly patients in whom the disease had actually begun many years before and had become arrested. With the onset of the senium and its disabilities the question of syphilis as a cause of the symptoms and the advisability of further antisymphilitic treatment will arise. It has been my experience that further antisymphilitic treatment is not indicated in such cases.

Senile Chorea. As a clinical pathologic entity this is questioned by some authorities who prefer to regard it as merely a symptom. The pathologic changes found in such cases are similar to those found in Huntington's chorea. It is a chronic condition for which no treatment is of value.

Senile Epilepsies. The so called senile epilepsies usually begin in the sixth or seventh decades of life. The attacks resemble the typical grand mal seizures, although they frequently show some of the characteristics of jacksonian seizures.

A patient of mine began having typical grand mal attacks at the age of

a day. Since a great many diseases of the brain may cause convulsive seizures, including such factors as transient vascular conditions, hypoglycemia, and others, a careful search for the cause is the prime requisite in treatment. Old people, as a rule, do not tolerate sedatives well and their use in these cases should

DISEASES OF THE SPINAL CORD

Senile Paraplegia This condition might better be called paraplegia in the senium in order to emphasize that different causes and pathologic states are included in this designation

The development of weakness in the lower limbs in advanced years is a common observation There is usually a mild spastic condition The legs feel stiff and heavy and the gait is slow, uncertain, and shuffling In spite of the spasticity, the tendon reflexes are diminished or lost and also the plantar reflexes Subjective sensory disturbances such as burning sensations, tingling, coldness etc., are common, but objective sensory changes are not severe Vibratory sense is more often affected than the other forms of sensation There is a general wasting, but localized atrophy is rare

The pathologic change in the spinal cord may be an arteriosclerosis or an annular degeneration around the periphery of the cord or, more often, both combined A rather characteristic pathologic finding in senile spinal cords is the presence of large numbers of amyloid bodies scattered through the white matter but more marked at the periphery Spiller¹¹ says, 'they are indicative of the senile changes which occur in every central nervous system' He also reports finding what he calls 'colloid bodies' that are slightly larger than the amyloid bodies and have different reactions He also describes "hyaloid bodies as evidence of senile degeneration A confusing feature of the pathology is the fact that quite often such changes are found in the spinal cord of aged persons in whom there have been no special symptoms In some of these cases the degenerative changes in the spinal cord resemble those seen in the spinal cord in association with pernicious anemia The axis cylinders are swollen or disappear in the lateral and posterior columns Injection of crude liver extract may cause decided improvement in the condition but it must be given in large doses

Chronic Infectious Myelitis A chronic type of infectious myelitis is not uncommon in the senium Usually the infection seems to originate in some part of the urinary tract, the so-called "urinary paraplegia" which was first described by Leyden in 1865 A long standing infection of the prostate or bladder is followed by a slowly developing flaccid paraplegia The weakness may be confined to certain groups of muscles A series of such cases was reported by Currier¹² It is supposed that the infection is carried to the spinal cord by way of the lymphatics The prognosis for recovery from the paralysis is generally poor, but many patients show decided improvement if the urinary infection can be eradicated Two of the patients reported by Currier were seventy two and seventy seven years old The seventy two year old patient recovered from the paralysis but died five months later of pneumonia A pathologic study of his spinal cord showed only the usual changes of senility

DISEASES AND INJURIES OF THE PERIPHERAL NERVES

Disease and injury of the individual peripheral nerves are as frequent in

readily than in a young person The symptoms and signs are similar but, as a rule, an injury or irritation of a nerve in an old person seems to cause less pain but more disability and is slower in recovery

Susceptibility to Toxins. Multiple neuritis, for various reasons, is quite common in the aged. The ordinary toxic agents causing multiple neuritis, such as alcohol, lead, arsenic, and so on, act not only more quickly but more severely. A patient of mine, seventy-two years old, applying the old fashioned preparation of lead water and laudanum to a leg ulcer, developed a typical lead neuritis. The various cachexias and nutritional disturbances of the aged may lead to the long-continued administration of "tonics" which eventually can have a toxic effect. It is said that prolonged administration of mineral oil will lead to loss of the fat soluble vitamins and cause a neuritis due to avitaminosis. Bieter and others,¹³ in discussing the toxic effects of sulfonamide derivatives on nerves, point out that definite changes of a degenerative nature occur with age and that possibly changes of this nature make a nerve more susceptible to the toxic neurotropic action of these drugs. These authors found that sulfathiazole can produce more nerve injury than sulfanilamide or sulfapyridine.

In 1905, C W Burr and C D Camp¹⁴ reported the effect of immobilization in causing a multiple neuritis. It was especially noticed in cases of hemiplegia where the elderly patient was confined to bed. Pain and a flaccid paralysis developed in the leg on the nonparalyzed side. At autopsy in these cases, a peripheral neuritis accompanying an endarteritis obliterans was found. I have seen this condition develop in elderly patients confined to bed for various reasons and its occurrence may be anticipated. The effect of the immobility can be diminished by encouraging the patient to move the part, also by passive exercises and gentle massage. Although degenerative changes apparently do occur in the peripheral nerves as the result of aging alone, I doubt if these would lead to serious disability unless aggravated by additional toxic or infectious states that are preventable.

Neuralgia. The most important type of neuralgia occurring in the senium is that affecting the trigeminal or fifth cranial nerve, sometimes called "tic douloureux." The cause of the condition is unknown. In younger people sources of irritation in the teeth or nose and throat may be to blame for a trifacial neuralgia and their removal stops the pain. In elderly patients this is rarely the case. It has been suggested that senile changes in the shape of the lower jaw may put the nerve on stretch but this is unlikely. Material removed at operation on the gasserian ganglion shows no significant changes. The condition usually begins rather insidiously with an occasional sharp "lightning" pain in the face. Gradually such twinges become more frequent and severe until the patient suffers agonizing attacks of pain in one or all of the three branches of the nerve. Talking, eating, or even the slightest touch to the skin may cause a paroxysm. Touching certain spots on the face, the so called "trigger zones," will bring on an attack, and this is a characteristic feature. In true cases, there are no clinical findings. There are no objective sensory disturbances and the motor innervation of the fifth nerve remains intact.

The prognosis for spontaneous recovery is poor. Many of these patients commit suicide or become drug addicts.

Medical treatment for the condition is usually unsatisfactory. Iron, quinine, strychnine, and arsenic in fairly large doses have been effective in some cases. I have also had good results with large doses of iodine.

The most effective treatment is surgical. If the nerve is injected with alcohol relief can be obtained, sometimes for months, but the pain recurs and

subsequent injections are less effective. Permanent cure is obtained by the surgical removal of the gasserian ganglion or by resection of its sensory root. Some years ago this operation had a high mortality rate but at the present time the rate is less than 1 per cent although many of the patients are over eighty years old.

Herpes Zoster The most common site for the skin lesions is on the thorax as a half girdle following the course of one or more of the intercostal nerves. Other areas are frequently affected however especially the distribution of the trigeminal nerve. The cause is a filtrable virus which affects the posterior spinal root ganglia. The appearance of the vesicles and the course of the disease is the same in both young and old. Usually the eruption disappears in the course of two or three weeks leaving scars that are permanent. If the vesicles are confluent large blisters may form (herpes bullosa) or the whole area may become gangrenous (herpes gangrenosa). The neuralgic pains may persist for a long time after the skin eruption has been cured and this is especially likely to occur in older patients. It has been assumed that the source of the pain was a persistent lesion in the posterior root ganglion. I have seen a patient however aged seventy four years who had a herpes zoster in the region of the tenth to twelfth dorsal root distributions on the left side which was followed by severe neuralgic pain in that region. Three months after the eruption a neurosurgeon did a laminectomy and cut the posterior spinal nerve roots on that side from the ninth dorsal to the first lumbar. There was a loss of sensation but no relief of the pain in that region. About three months after that he did a unilateral cordotomy in the upper thoracic region. Following this there was a loss of pain and temperature sense on the left side below the level of the operation but no relief of the neuralgia.

In herpes zoster ophthalmicus the scars on the cornea may cause marked impairment of vision.

Herpes zoster is rarely recurrent.

The acute eruption requires no special treatment except the protection of the lesion by the application of carbolated petroleum jelly on gauze. The relief of the neuralgic pain is difficult and it may persist for years. Local applications are generally useless. Injections of pituitary gland extract have been used with some success especially the extract of the posterior pituitary. Recently there have been reports that injections of large doses of vitamin B have given relief to these patients (see p. 740).

Functional Disturbances It must not be forgotten that emotional disturbances are quite common in aged persons and that these phenomena may complicate the clinical picture in any of the above conditions. I have seen

electricity) brought about an almost complete return of function after one treatment.

SUMMARY

To summarize it may be said that while the basic factor in the cause of the degenerative diseases of the nervous system in old age probably is the playing out of the inherent vitality of the cell structure other factors are im-

CHAPTER 19

DISEASES OF THE EYE

BENJAMIN RONES

INTRODUCTION

THE eye and its adnexa offers a very favorable organ in which to observe senile changes both physiologic and pathologic. The organ is so constructed and situated that it is accessible both to direct and ophthalmoscopic examination, and its functional derangements can be measured accurately and correlated to its observed disturbances.

All the tissues of the eye are subject to changes with advancing age. In some ocular structures these variations manifest themselves by alterations in function, in others by visible degenerative disturbances, while other tissue changes can be seen only on microscopic examination. It is often difficult to differentiate these senile variations from pathologic lesions, for numerous diseases of the eye are most common in those years when senile changes occur. It must be borne in mind that the relationship between tissue age and chronologic age is not an exact one. Some individuals have advanced senile changes in their ocular tissues at forty years of age, while others have singularly little evidence of these at eighty. Nevertheless, in every individual past the age of forty years the ocular tissues will show senile alterations although these may be only in the initial stages. The boundary between the normal and the senile is also very difficult to define as exemplified in the continued growth of the lens throughout life.

EXTERNAL CHANGES

The Skin and Conjunctiva In the elderly the skin of the lids becomes thin and wrinkled due to the disappearance of the underlying fat. Such "crow's feet" are especially prominent at the outer angles of the eyes. Folds and pouches are formed in the lower lids, owing to a loss of the elastic fibers. The variations in the tone of the musculature and the loss of skin elasticity will produce the ectropion or entropion so commonly seen in the elderly. In women flat yellowish patches, xanthelasma, frequently develop along the inner angles of the lids, apparently as a result of a disturbance of the cholesterol metabolism. Also, owing to a diminution of the orbital fat, enophthalmos develops so that the eyes have a sunken look.

In the conjunctiva the most common senile change is the *pinguecula*, which appears as a yellowish slightly elevated spot, commonly on the nasal side of the bulbus. It is usually situated close to the limbus and rather firmly

attached to the sclera. The re-
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Cornea. The cornea exhibits the most striking of all senile changes in the eyes—the *arcus senilis*. Clinically, it is seen as a zone of opacity around the circumference of the cornea, separated from the margin by a narrow band of comparatively clear tissue, and showing a sharply defined edge on this side. The edge towards the center is much more indefinite and shades off gradually into the clear tissue. Although the advanced arcus encompasses the entire circumference of the cornea, in its earlier stages it is usually present as an opaque zone above and below, each of which extends laterally until they unite to form the complete ring. Such a ring is at times seen in youth and is then called a *juvenile arcus*. On microscopic examination the cornea is found to be infiltrated with fat globules, in such a manner as to produce the clinical picture



Fig. 64 Arcus senilis. This fat stain shows Bowman's membrane to be heavily laden with fat. The point of termination of this membrane is well seen. In the corneal stroma the greatest concentration of fat is beneath Bowman's membrane and in front of Descemet's. The latter is also deeply infiltrated (Rones. *Am J Ophthalmol* 21.)

Small warty hyaline excrescences on Descemet's membrane are a frequent finding when the cornea of aged individuals is examined with the corneal microscope. Of such

progressive opacification of the cornea results. An increasing loss of vision results from this later *epithelial dystrophy* and no treatment has been found to be of any value in slowing or preventing this progression.

The Iris. Since the advent of the corneal microscope, the senile changes in the iris have been subjected to considerable study. A very common finding is the disappearance of the pigment at the pupillary margin, leaving a whitish and ill defined border having a hyalinized appearance. Again, due to an in-

crease in the connective tissue and hyalinization of the septa of the sphincter muscle, the pupil becomes smaller and does not react actively. This *senile miosis* and rigidity of the pupil must be differentiated from the abnormal pupillary changes of disease. The entire iris becomes thinned and flattened so that many of the crypts disappear from its anterior surface. At times the atrophy of the stroma is of a patchy distribution exposing the underlying pigment epithelium. This pigment layer regularly shows some degree of depigmentation in old age. The scattered pigment granules released by these cells are then found on the posterior surface of the cornea, the anterior surface of the iris and lens capsule, and enmeshed in the trabeculae of the anterior chamber angle. The arteries of the iris show marked thickening and hyalinization of their walls as a characteristic senile change.

OPHTHALMOSCOPIC VARIATIONS

The Vitreous. A frequent complaint of elderly patients is the appearance of floating spots before the eyes. Examination usually shows stringy *vitreous opacities* which move fairly rapidly with the excursions of the eye. No inflammatory lesions are visible, and the condition does not progress nor is the visual acuity diminished. The opacities are due to a senile disintegration of the fibrous network of the vitreous so that this body loses its gel characteristics and becomes more fluid. At times cholesterol crystals, the so-called *'synchysis scintillans,'* will be deposited in such a degenerated vitreous. These highly refractile particles floating in the vitreous afford a very dramatic ophthalmoscopic spectacle but cause no visual disturbance. At other times the floating bodies resemble cotton balls due to the deposition of calcium soaps.

The Retina. In children and young adults the retina has a glistening transparency when viewed with the ophthalmoscope and there are many dancing light reflexes on its inner surface. With advancing age, however, the retina becomes less transparent and has a duller appearance. *Senile macular degeneration* is a very common finding in the elderly. The chief complaint of these patients is impaired central vision, and although they are able to get around and carry on their general activities, the ability to read and perform the finer visual tasks is considerably diminished. Ophthalmoscopically, the condition is difficult to detect in its earliest stages, but careful examination will reveal a pigmented stippling in the macular region. Although only one eye is involved initially, it is invariable that the other eye becomes affected after some time. With the slow advance of the condition, the pigment becomes heaped up in the macular zone, and adjacent whitish patches of atrophy develop. The resultant central scotoma is permanent.

The development of colloid bodies or *drusen* on Bruch's membrane is a common senile manifestation. Ophthalmoscopically these are small, whitish, sharply circumscribed and slightly elevated spots beneath the retina. At first they are discrete and scattered over the posterior pole of the fundus, but later they enlarge and coalesce, often forming rather large masses in the macular zone. They usually cause very little visual disturbance, although when the overlying retinal and choro-

roidal vessels

Sclerotic changes in the retinal blood vessels have been studied exhaustively.

tively, both clinically and pathologically. Correlation of the evidence produced by these methods of investigation has not been entirely satisfactory, however. A differentiation can be drawn between arteriosclerosis and arteriolarsclerosis, for the two types are quite distinct pathologically. When the arterioles of the body are involved in the diffuse type of hyalinization of the media, diverse hemorrhages and exudates are found in the retina as ocular complications, while the general body resultants are hypertension and cardiorenal impairment. This type of sclerosis is seen with greater frequency in middle age than



Fig. 65 Arteriosclerosis in the choroid (Rones. *Am J Ophthalmol*, 21)

in the elderly. Generalized arteriosclerosis, with a thickening and lipoidal infiltration of the intima, is observed in elderly persons usually without an associated hypertension. Friedenwald has described the ophthalmoscopic picture, stating that the larger retinal vessels are of normal caliber, but show beading, irregularity in the light reflex, visible walls, and very rarely arterio-venous compression, hemorrhages, or exudates, the arterioles are normal. In many old people, however, a gradual rise of blood pressure occurs, usually less pronounced than in the younger age group. When this complicating factor of hypertension occurs in the generalized arteriosclerosis of the aged, the most

characteristic change is a narrowing and straightening of the retinal arteries; also the vessel walls become more visible, and consequently their light reflex is more pronounced. Compression of the veins at the arteriovenous crossings becomes marked, and later the picture of arteriosclerotic retinitis, with numerous hemorrhages and exudates, develops.

The Choroid. Such sclerotic changes of the vessels are also the chief senile variation in the choroid. Lipoidal infiltration of the intima and muscularis causes a narrowing, or even total occlusion, of the lumen of the arteries. These changes occur in the larger arteries, causing an atrophy of the capillary bed which they supply. Since all of the arteries are not affected, this atrophy of the choriocapillaris is patchy in distribution, while in the adjacent areas a compensatory dilation of the capillaries will take place. These variations are most marked in the posterior pole of the eye and are readily visible ophthalmoscopically. The occlusion in the choriocapillaris will cause impairment of the retinal pigment and outer retinal layers, which are dependent upon the capillary circulation of the choroid for nourishment. Thus there arise such conditions as senile macular degeneration and drusen formation, as previously mentioned.

The Optic Nerve. In the optic nerve arteriosclerotic changes occur particularly in the small nutrient arteries arising from the pia-arachnoid sheath and penetrating into the nerve from the periphery. The resultant atrophy may be patchy, or may progress to complete destruction of the nerve and blindness. Visual field defects will occur, corresponding to the damaged bundles of nerve tissue. Attempts have been made to improve such conditions by the retrobulbar injection of vasodilator drugs, but results have been conflicting. A favorite site for the formation of atheromatous plaques is in the central artery of the nerve just as it enters the eyeball. This is a common predisposing cause of thrombosis in the retinal vessels, both arteries and veins. It should also be borne in mind that one cause of optic atrophy in the elderly is the pressure of a sclerotic internal carotid artery upon the nerve in the region of the chiasm. When this occurs on both sides of the chiasm, the diagnosis is easily made by visual field studies.

FUNCTIONAL VARIATIONS

Presbyopia. In the fourth decade of life the eye begins to lose its ability to accommodate for close work. This condition, termed *presbyopia*, does not affect distance vision but requires that reading material be held farther away from the eyes. With the recession of this near point beyond the range of comfort it becomes necessary to correct the close vision by wearing convex glasses.

As a result of the changes of age, it may be said that the factors that determine the demands made upon the eye for close work, the general physical health of the person, and particularly the hereditary factor of longevity. Presbyopia develops as a result of the changes in the lens.

The lens is formed in size from birth until death. This occurs by the constant apposition of new lens fibers. The volume of the lens is held within definite limits, how-

ever, for the newly formed peripheral fibers constantly compress the central nuclear mass. This sclerosis, resulting from a water loss and chemical change, diminishes the elasticity of the lens and consequently prevents it from assuming the more convex shape required for focus at close range. Also, since the lens increases its density by this loss of water content, an artificial myopia frequently results due to the increased index of refraction. This phenomenon is spoken of as "second sight" and the elderly individuals in whom it occurs are again able to read without glasses but may require a myopic correction for distance vision. Such lenticular alterations are physiologic as long as the tissue remains transparent and vision is not affected.

The sclerosis of the lens plays the major role in the production of presbyopia. Infiltration of lipid into the ciliary muscle diminishes its activity and consequently weakens the accommodative effort, however. With age there is also an elongation of the ciliary processes and an increase in their bulk, pushing the root of the iris forward and partially occluding the filtration angle. It

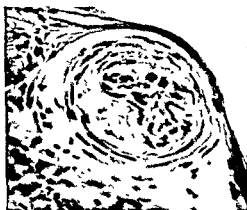


Fig. 66 Sclerosis of the central retinal artery on the papilla. The proliferated endothelium has almost occluded the lumen (Rones. *Am J Ophthalmol*, 21)

has been frequently stated that this is the precipitating factor in the production of glaucoma, but it certainly is not the only cause.

The Visual Fields. In elderly people contracture of the visual fields should be interpreted cautiously. A concentric contraction of the fields is frequently found. The cause of this is obvious when many elderly eyes are studied histologically. A common senile change is an atrophy or a cystic degeneration of the periphery of the retina. Apparently this results from the diminished circulation through the sclerosed vessels. Sector-like defects in the visual field are also seen at times. Such changes are due to a corresponding sector atrophy of the optic nerve and, as mentioned above, result from sclerotic changes in the nutrient vessels of the nerve.

CATARACT AND GLAUCOMA

These two conditions cannot be classified among the physiologic changes of senility. They must be given consideration here, however, since they occur predominantly in the age groups under discussion.

Cataract. Although cataract can result from a number of general and local diseases, and from injury, exogenous toxins, radiation, etc., it is a condition primarily associated with senility. The lens is of ectodermal origin, and its manifestations of senescence are similar to those in such epithelial structures as the skin, hair, and nails. Essentially this is a process of gradual dehydration and decreased metabolism, with secondary necrosis of tissue. The normal aging lens remains transparent, although its density increases by the compression of its nucleus and although a grayish or yellowish discoloration may occur. Frequently, however, careful examination with the ophthalmoscope and corneal microscope will reveal vacuoles, clefts, and separation of the lens sutures, and exfoliation of the anterior capsule. Such changes usually precede the coagulative necrosis of lens fibers giving rise to the opacity in the lens, termed cataract. It can be stated that such lenticular opacities are the rule in advanced age, but since the majority of them are peripherally located and very slowly progressive, they do not interfere with vision and the patient is consequently unaware of any disturbance.

When the opacities radiate in towards the center of the lens, however, vision does become impaired, and its subsequent loss will depend on how fast the opacity develops throughout the lens. In the nuclear type of cataract, the opacity first manifests itself in the center of the lens. This is really an advanced stage of the physiologic state of nuclear sclerosis. Such lenses frequently have a dark brown or black discoloration, rather than the whitish appearance of the typical "mature" cataract. In making the diagnosis of senile cataract, great care should be taken to rule out the possibility of an accompanying glaucoma or an inflammation of the uveal tissues, for these will materially affect the prognosis. The function of the eye should be tested at regular intervals to determine the progression of the opacity.

No therapeutic measures have been found to be of any value to clear a lens opacity, or even to retard its progress. Many methods have been advocated, recently vitamin C and riboflavin have been extolled for this purpose. Although these vitamins have been found to play a role in the metabolism of the lens, it is extremely unlikely that they can reverse coagulative necrosis which has already occurred. Our only therapeutic method for a mature cataract still remains the surgical removal of the opaque lens. Nevertheless, in the earlier stages, everything possible should be done to promote the general health of the patient, and in that way the nutrition of the lens. Particular care should be given to avoid the use of the term "cataract" during these early stages, for from long usage this term has come to connote blindness to many individuals. It should be remembered that these lens opacities often progress so slowly over a period of years that death intervenes before the individual is aware that there is anything wrong with his eyes.

Glaucoma. Glaucoma is a much more serious affair than cataract, for

this time. The patients do not complain of any visual disturbance, and only

tion of the visual field occurs until it assumes a gun-barrel shape in --

vanced cases. The central vision usually remains unimpaired until very late. The condition is essentially an increase of the intraocular pressure, so that a pressure atrophy of the optic nerve slowly takes place, manifesting itself by a gradually enlarged cupping of the disk. This damage to the nerve can be measured in the enlargement of the blind spot and the appearance of scotoma. The pressure within the eye is measured with a tonometer. In the earliest stages there is a fluctuation of pressure, so that repeated measurements should be taken at various times of the day in order to detect an elevation. If the condition is not detected and treated, congestive attacks will cause great pain and, later, total blindness.

If glaucoma is recognized early, the use of miotics may control the pressure for many years and prevent the progression of the disease. Supportive measures such as rest, emotional stability, diet and other such general therapeutic means should also be applied. If the intraocular pressure cannot be controlled by such medical procedures, surgical measures become necessary. These are successful in checking the disease in a high percentage of cases.

Glaucoma is a condition that can be checked, but whatever vision has been lost can never be restored. It must be stressed, therefore, that the sooner it is recognized and proper therapy instituted, the greater the amount of vision that will be saved. Many theories have been propounded as to the cause of this disease, but the true explanation is still elusive. Although it would seem that it is always associated with a disturbance of the capillaries, involving stasis, a definite relation to elevated blood pressure cannot be established. Chronic simple glaucoma is probably not a single disease, but rather it appears to be a variety of entities having the common factor of an increased intraocular pressure.

TUMORS

Melanoma of the Uveal Tract. The varieties of tumors occurring in the eye depend upon the age incidence, as they do in other parts of the body. The commonest ocular tumors after fifty years of age are the malignant melanomas of the uveal tract, most frequently originating in the choroid. Clinically such tumors have been divided into four stages: (1) a symptomless stage, (2) a stage of glaucoma, (3) extraocular extension, (4) generalized metastases. Generalized metastases, especially to the liver, can occur at any stage, however, even before the ocular tumor has been detected. If the macular region is involved, visual disturbance will be an early symptom. Often there will be no warning until the tumor has grown enough to produce a partial detachment of the retina. The melanomas are a malignant type of tumor, eventuating in a high percentage of deaths unless the diagnosis is made very early. Removal of the eye is the only treatment for this condition, and the sooner this is performed, the better the chance that metastasis has not already occurred. It is not always an easy matter to make an early positive diagnosis, for such confirming signs as retinal detachment, increased intraocular pressure, and poor transillumination may all be absent.

Metastases to the Choroid. The choroid is also the site of metastatic tumor growth, particularly from carcinomata originating in the breast, and less frequently from the lungs, thyroid, or stomach. Cases have been reported where such metastatic growths were found in the choroid before the primary tumor was detected.

SUMMARY

In reviewing the changes that occur in the eye during old age, it is obvious that in this organ, just as in other parts of the body, it is impossible to draw a line of demarcation between the physiologic processes of senility and pathologic degeneration. It is also manifest that senile changes do not occur only in the aged, but often begin in those quite young and form a continuous process. The problem of senility should rather be looked upon as the wear and tear of tissues due to the stresses and strains of life, influenced considerably by hereditary factors.

The aging eye steadily loses some of its functional efficiency. Everything that contributes to the general welfare of the individual will delay this slight but steady loss. Frequent observation will also allow the diseases that occur in the aged to be detected early and proper therapy instituted.

CHAPTER 20

DISEASES OF THE EAR

G HAVEN MANKIN

SENESCENCE is physiologic or normal aging. We must keep in mind the changes attributable to normal aging, otherwise we cannot draw the line between senescence and senility and the diseases associated with advanced age. Today we accept many of the changes of advancing years as an integral part of the ravages of time. I have in mind loss of teeth and hair, whitening of the hair, dryness of skin, impairment of sight and hearing, and the arcus senilis, to mention a few. It is quite possible, that in the future, through preventive medicine, proper hygiene, and diet control, particularly a better understanding of the role of vitamins, these changes will not necessarily constitute an essential part of senescence.

Impairment of hearing produces definite psychologic changes in the individual. He becomes suspicious and feels he is being left out of conversations and discussions, and may even become resentful because he imagines he is being talked about. Rarely is the psychologic reaction so noticeable in a person with impaired vision. Such an individual is usually cheerful and happy, and does not exhibit a suspicious nature.

Deafness in the aged is not necessarily a component part of the normal, physiologic aging process, although physicians are prone to consider it to be. We can all recall friends, relatives, and patients with serviceable hearing even at the age of ninety.

If an audiogram were made from the "normal" aged patient it is likely that there would be noted a definite loss of the higher tones. Unless the loss extended down into the conversational range (32 to 2500 double vibrations per second) no subjective impairment would be noted. Patients often seek the aid of the otologist because they cannot hear the tick of a modern wrist watch. It must be remembered that the tick of a wrist watch is well above the conversational range and is therefore a poor test of a patient's ability to hear conversation.

PERCEPTIVE DEAFNESS

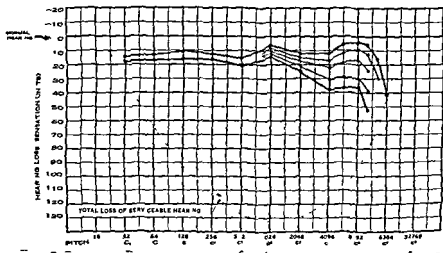
This condition is usually found in some degree in most individuals over sixty-five years of age. When uncomplicated by previous or present middle ear disease, it is characterized by high tone loss, decreased bone conduction, and a positive Rinne reaction. The individual thus affected seldom has disturbing difficulty in hearing ordinary conversation with one person, but he is very much handicapped in hearing clearly a discussion in a conference or where there is a background of voices.

Bunch and Raiford¹ tested the hearing of a large group of patients at the Johns Hopkins Hospital, who had no complaint of ear trouble or of hearing loss. The age groups ranged from twenty to twenty nine in one group, and over sixty years of age in the other group. The lower age group showed very

little hearing loss until 8192 double vibrations per second was reached, as indicated by a standard audiometer that produced tones with frequencies of from 32 to 16,384 double vibrations per second. The older group, however, began to show a gradual hearing loss above 1024 double vibrations per second and a marked loss above 8192 double vibrations per second (See Fig 67)

We have been discussing hearing by air conduction, the usual manner in which conversation is heard, and have not mentioned hearing by bone conduction. We must consider the latter, however, if we are to interpret tuning fork tests and hearing of telephone conversation, and if we are to be helpful in advising the patient in hearing aid selection.

Struycken² felt that the upper limit of hearing by bone conduction regresses with age more or less parallel to the upper limit by air conduction. Complete accuracy in making these tests is difficult of attainment. Most otolo-



ment for each decade

gists use the C-512 fork in determining the bone conduction of an ear, and in carrying out the Rinne and Weber tests. This fork is well within the conversational range, but it has the disadvantage common to all tuning forks, namely, difficulty in standardizing the intensity of the tone. One is often struck by the observation that an older individual with serviceable hearing for ordinary conversation may have a rather markedly decreased bone conduction when tested with the C-512 tuning fork, and by the bone conduction attachment of the audiometer.

Guild³ believes that the osseous tissue of the ear, like other bones in the

of the trabeculae of the floor of the aditus ad antrum. These fractures were considered to be spontaneous. The clinical examination had indicated im

paired hearing by bone conduction. He suggests that sound waves transmitted through the bony trabeculae of the floor of the aditus ad antrum reach the fluids of the inner ear from the direction best for effective stimulation of the organ of Corti.

Crowe, Guild, and Polvogt,⁴ through examination of temporal bones of patients who had a careful otologic and hearing test prior to death, found a

was due to anoxemia secondary to the vascular changes often noted in the aging individual must be answered in the future.

THE MIDDLE EAR

When the middle ear is the site of pathologic change in an aging individual, he is likely to show the symptoms and signs of conductive deafness, in addition to those of perceptive deafness, discussed in previous paragraphs. Every effort must be made to correct the pathologic condition in the middle ear. This often entails the correction of defects in adjacent structures, such as the sinuses, nasopharynx, and throat. It is obviously impossible to return to normal function a middle ear which is full of adhesions that restrict the movement of the tympanic membrane, the ossicles, the tensor tympani, and the stapedius muscles. One can, however, take steps to clear up active infection in the middle ear and prevent, to a large extent, further insults to that area by

nasopharynx. The nasopharynx must be inspected with a nasopharyngoscope to see if the pharyngeal orifices of the eustachian tube are obstructed by infected lymphoid tissue. Often the eustachian tube is obstructed by swollen and infected lymphoid tissue within the lumen of the tube itself. This tissue can be eliminated by irradiation.

It is trite to say that many of the hearing disorders of the aged really begin in childhood, but it is certain that, if we are going to prevent with any degree of success the impairment of hearing due to poor conduction, we must begin when the patient is a child. In adults, the defect has been many years in the making and irreparable damage to the hearing apparatus has already been done.

Radiation Treatment. Recognizing this, Crowe and Burnam⁵ have standardized the use of radon and radium in the nasopharynx, and have emphasized the necessity for reduction of upper respiratory infection in children, if middle ear involvement is to be avoided. This procedure is more effective in children than in adults, but adults should not be denied its benefits if there is evidence of nasopharyngeal and eustachian tube infection.

Indiscriminate use of radium as a treatment for deafness is condemned, but radiation is of definite value in reestablishing the patency of a eustachian tube which has been blocked by swollen and chronically infected lymphoid tissue. Its use is justified in all forms of deafness from whatever cause, in order to prevent superimposed or additional impairment due to lymphoid hyperplasia in and around the eustachian tubes. A frequent check of the condition of the nasopharynx must be made by the electric nasopharyngoscope.

Lymphoid tissue is extremely sensitive to irradiation; mitosis and cellular division do not take place, and cellular growth diminishes. By careful determination of the dosage required, only the lymphoid tissue is affected, and no other structures are involved.

The most convenient form of radiation of the nasopharynx is the use of the radium nasopharyngeal applicator, which consists of a Monel metal capsule in which the radium element is sealed, a brass wire stem, and a lightweight aluminum handle. Due to the small size of the capsule (outside diameter = 2.3 mm), it can be passed by almost any nasal obstruction. The capsule contains 50 mg of radium sulfate, standardized by the manufacturer and the National Bureau of Standards.

Such an applicator is passed under local anesthesia along the floor of the nose to the nasopharynx and held in position for twelve minutes by scotch tape. This is carried out on each side of the nasopharynx. No discomfort is noted by the patient. There is no sensation, except the awareness of the presence of an applicator at the back of the nose.

With this method, the patient requires two to five treatments at intervals of two to three weeks. Effects are delayed and usually are not demonstrable until four or six weeks or longer have passed.

Treatments should not be given during an acute upper respiratory infection, as there is always the chance that an acute otitis media may result.

A new series of treatments should not be begun until ample time has elapsed during which changes may be observed in the nasopharynx and in the patient's hearing (six months). The radium applicator is a relatively new instrument in our treatment of deafness. It should be used with caution, and we should benefit by the experience of those who have used it safely over a long period of time. At the present time, it can be said that the dosage given above is perfectly safe. It is quite possible that experience may indicate that a larger dosage is entirely safe, but for the present we must be conservative and must be guided at all times by physicists and radiologists who are experts in this field. So far, no instances of dry nasopharynx or damage to adjacent structures following use of the applicator have been reported. Perlman⁶ used massive irradiation in treatment of carcinoma of the nasopharynx and found that conductive deafness due to carcinoma with resulting tubal obstruction and exudative catarrh can be completely relieved without injury to the peripheral auditory apparatus.

HEARING AIDS

Where hearing loss within the conversational range is greater than 25 per cent, the aging patient will need a hearing aid, usually of the air conduction

hearing against an improvement which may be of temporary nature, or against a chance of total deafness if labyrinthitis occurs as a result of the operation

Therefore, a properly fitted hearing aid holds the only definite and completely safe help for the hard of hearing, aging patient. The hearing aid must be selected with utmost care, after the most searching and thorough examination by a competent otologist to determine the best type of instrument for the individual patient

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SECTION IV

DISORDERS OF THE RESPIRATORY SYSTEM

CHAPTER 21

DISEASES OF THE UPPER RESPIRATORY TRACT

ARTHUR W. PROETZ

THE changes in the character of the upper respiratory tract due to old age are likely to be much less upsetting and disabling than those encountered in some other regions and organs. They are more likely to cause annoyances and discomforts than real disabilities and rarely have any effect upon life expectancy.

The membranes of this tract are exposed to the changes and vicissitudes of the environment even more than the skin. Not only are they unprotected by clothing but in the nature of their function the air with its contaminations is actually drawn over them and brought forcibly into contact with them in large quantities.

Many an argument has been waged over what constitutes a normal adult nose, since the effects of irritation may be said to leave their mark in practically all individuals who have reached adult life. To state that there is no such thing as a normal nose in adults is merely to cavil with the definition of normality. Noses of adults all show some departure from the original pattern, but it is useful for purposes of prophylaxis and treatment to differentiate between the changes in mucosa and musculature resulting purely from old age and those resulting from extraneous influences operating through long periods of the subject's early and middle years.

One need scarcely call attention to the fact that the state of nutrition and physiologic activity in a nose must be an inseparable part of the general condition of the individual. In a person, old or young, who is below weight from any cause, whose cheeks are sunken, whose neck is thin, whose hands are emaciated, one expects and finds similar changes in the nasal alae, in the nasal conchae and the membranes in general, and also in the pharynx. These are conditions upon which local treatment has obviously no effect. In a younger person the underlying cause of such atrophies and malfunctions can usually be found in some systemic disorder and the local conditions may be expected to improve when the systemic defect is corrected. In the aged, however, in whom rehabilitation of the vital mechanisms is not possible, the problem becomes one rather of adaptation than of repair. Therefore our first duty is to determine whether in a given case the symptomatology is the result of (1) the wear and tear of environment, (2) a systemic disorder capable of repair, or (3)

of middle life may assume the proportions of a major disturbance to tranquillity in old age. With our growing consciousness of the geriatric phases of medicine it will therefore become our responsibility to give more attention during middle life to those disorders which may result in such disturbances later on.

The affections of the nose and throat which can be strictly classified as the concomitants of age are few and result largely from simple atrophies of the mucosa, the glands, and the muscles, similar to those found elsewhere in the body.

EXTRINSIC CAUSATIVE AGENTS

Among the environmental agencies with which we are chiefly concerned are weather, infections, systemic diseases, operations, smoking, exposures to industrial dusts and fumes and overheated living quarters.

While persons spending their lives in rural communities show few changes resulting from mere breathing, this is not so with the city dweller. Throughout life he is exposed day and night to such corrosive substances as sulfur dioxide and trioxide and various tars, the incomplete combustion products of coal, exhaust gases from motors, the dust of city streets, and particulate matter in endless variety from manufacturing plants.

Since the development of air and automotive travel, permitting a constant influx of population from distant communities, the city dweller especially is exposed to bacteria and viruses of many types and strains which are new to him and to which he has little immunity. Each passing irritation and infection contributes its small quota of fibroblasts, eventuating ultimately in generalized fibrosis and later atrophy.

While many local infections may not properly be considered as contributing to the derangements of old age, common colds are universal in their distribution and play a large part in the hyperplasias of middle life and the atrophies of old age, as will be shown.

Patients now in advancing years spent their prime in the early decades of the present century and many were caught in the wave of nasal surgery which swept Europe and America in that day. The removal of comparatively large areas of essential structures which was then freely practiced is the cause of the atrophic states from which they still suffer.

These atrophies may be generalized or localized depending upon the amount of tissue removed and upon the relationship of the remaining struc-

include the openings of the eustachian tubes there may be interference with hearing, due either to closure by swelling or to hyperpatency by wasting. These effects will be further discussed.

cially of the velum palati and the uvula. This is especially pronounced in pipe smokers. The condition may or may not extend to the larynx and to the nose, and this is determined by the practice of inhaling or of blowing the smoke through the nose.

Tobacco smoke consists largely of very minute globules of tarry substances held in suspension. So long as smoke flows evenly without restraint and in a relatively straight pathway, very few of the suspended particles come in contact with the walls to be deposited there. Sudden deflections in the course of the air current and, even more especially, constrictions in the channel produce eddies forcing the droplets into contact with the membrane and depositing them. Thus in the noses of smokers, tar deposits and their effects can usually be found just anterior to some constriction or obstruction such as a septal spur, a hyperplasia, or an adhesion. Years of smoking at first bring about the vascular dilatation described, which in old age eventuates in fibrosis, ischemia, and atrophy.

Industrial smoke, dusts, and fumes have much the same effect except that they are inhaled and the involved area is proximal instead of distal to the obstruction. The rapidity with which they work depends upon their nature. Sulfur dioxide and sulfur trioxide in contact with the nasal moisture form sulfurous and sulfuric acid. These gases are among the most corrosive of the components of ordinary soft coal smoke. Exposure to heat alone in bakeries, smelters, and foundries is likely to produce only a chronic hyperemia which rarely goes on to the stage of atrophy.

The list of extrinsic agencies is not complete without mentioning, indeed underscoring, the *overheating of living quarters* in winter. Americans are prone to heat their houses excessively. It is not only the vascular response to the heating which is undesirable but the excessive dryness which accompanies it. In winter the air outdoors contains very little moisture. When this dry air is brought indoors and heated the relative humidity sinks to a very low level. The air gathers moisture wherever it can get it, and thus dries the nasal mucosa unduly.

With the loss of moisture the mucus becomes viscid and finally dry, the cilia become inactive for long periods of time, minor infections are the rule, and the end result is rhinitis sicca, a very irregular hyperplasia, or an atrophy.

Corrective Measures While these conditions cannot be repaired in the aged the symptoms referable to them can be alleviated to a large extent by *humidifying the living quarters*, and, in particular the sleeping rooms. Much more water is required to bring winter air to the desirable relative humidity (40 per cent) than can be supplied by open vessels of water set about the room. Keeping the tea kettle going, allowing the shower to run with the bathroom door open, or filling the tub with hot water are effective, but have the disadvantage that the steam is partly condensed on nearby objects or occasionally upon the clothing.

Much better are the various devices which emit a spray of finely divided cold water. These are *not expensive and are quite satisfactory*.

Oil mixtures are ineffective in overcoming this dryness, and should not be used. Their selection is illogical, what the nose requires is moistening, not lubrication. The inspissated secretions are not soluble in the oil, which only

covers the surface and renders the glands even more ineffective through lack of stimulation

Simple *water sprays* would seem indicated but the effect of these is only temporary, as they are rapidly evaporated in the process of respiration. While there is no wholly satisfactory treatment, very good results may be obtained through the use of a mixture of ethyl alcohol (5 per cent), glycerin (3 per cent), and physiologic sodium chloride solution (92 per cent). This serves to stimulate the glands and moisten the membrane and at the same time retains the moisture temporarily. The mixture has a mildly unpleasant odor and may be improved by the addition of a drop or two of some harmless scent such as spirits eau de cologne.

The administration of *endocrine substances*, as has been recommended for atrophic rhinitis, has not proved effective.

EFFECTS OF INFLAMMATION ON THE NASAL MUCOSA

Before proceeding to the specific changes which occur in the various portions of the upper respiratory tract it may be profitable to digress briefly and to consider the effects of inflammation upon the nasal mucosa. Basically these differ in no wise from inflammatory reactions elsewhere in the body, the special symptoms which they produce are rather the result of the mechanical disposal and arrangement of the nasal structures.

In the beginning, inflammatory processes are vascular. a transient ischemia is followed promptly by an arterial hyperemia. This is especially noticeable in the nose owing to its great vascularity. As in other engorged tissues venous stasis soon follows, the nose remains obstructed, the color of the membrane changes from a bright vermilion to a deep purple, glandular activity is upset, and exudation begins.

The loose reticular tissue of the tunica propria is distended with cellular elements. If the inflammation is of short duration few of these remain and with resolution the tissues return to practically their previous state.

If the inflammation is protracted there is always some proliferation of fibroblasts, resulting ultimately in hyperplasia. The fibrosis occurs first about the arteries. In the course of time the fibrous tissue contracts, and by irregularly constricting the vessels roughens the mucosal surface. This produces a mottling in the color of the surface. At this stage the turbinates are thickened and offer a leathery resistance to the probe. The airways are still encroached upon. Finally the process ends in a generalized atrophy of all the elements of the mucosa. This is the condition which we are apt to encounter in the aged. Now the membrane is gray, thin, and dry, owing to the very restricted circulation and the consequent failure of glandular function. Secretions are scant and the airways are wide. Certain of the muscles, notably those of the pharynx and the larynx, participate in the general atrophy. The reaction at this stage is of course irreversible and hence incurable. It does not follow, however, that the symptoms cannot be greatly relieved. The symptoms which accompany the foregoing series of events are largely dependent upon the location.

The attention which is currently being given to proper environment and air conditioning will no doubt result in healthier noses, hence in a diminution of the late effects of continued irritation and infection.

THE EXTERNAL NOSE

Collapse of the Alae Nasi When this condition arises in old age, it is usually the result of inadequate alar cartilages, the lack of support narrowing the nostrils. In earlier life the activity of portions of the quadratus labii superioris muscle may largely compensate for this. A weakening of the muscle later on prevents the dilatation of the nostril, which collapses under the impact of forced inspiration. Drops of moisture may collect about such an opening. This and the collapse itself on deep inspiration are the only symptoms.

Treatment consists in holding the alae away from the septum by means of suitable wire splints or in surgically diverting triangular pieces of cartilage laterally from the septum to support them.

Rhinophyma This is a chronic overgrowth of the skin of the tip and alae of the nose eventuating in a bulbous deformity. It is occasionally met with in earlier life, but since it is the end result of an acne rosacea of long duration, it seldom occurs until middle age. It is often, though not always, associated with habitual liberal use of alcohol and is said to accompany seborrhea of the scalp. It has also been thought to be related to thyroid hypertrophy.

The mass is essentially a hyperplasia of the skin, induced by persistent hypernutrition with dilatation, and sometimes pustular infection, of the sebaceous glands. It is characterized by a scattered scarring which produces a pebbled of the surface. Owing to impeded circulation the mass assumes a deep purplish color.

Treatment consists in the surgical removal of the excess skin, which reduces the nose to something like its normal proportions. Granulation must not be controlled until epithelization has taken place. Skin grafts may be necessary.

Nasal Fissure A very troublesome complaint, found especially in under nourished individuals, is fissure of the external naris. This is usually the accompaniment or the sequela of chronic inflammation. It may occur anywhere in the nostril but is usually at one or the other extremity where the skin is folded upon itself. It may be very painful and render the tip of the nose sensitive to the touch. The treatment consists first in the control of any acrid secretions which may come down from the nose. After this the fissure should be spread wide open, local anesthesia being used if necessary, and vigorously freed of diseased skin by rubbing first with moist, then dry cotton pledgets.

collapsible tubes

THE INTERNAL NOSE

Nasal Passages As the condition of the mucosa and especially the tunica propria depends largely upon the state of the vascular system, and as this latter is subjected in the nose to much abuse in the course of a lifetime, circulatory and hence nutritional disturbances are common. These are ordinarily not severe but manifest themselves only in nasal pallor and overventilation, or in rhinitis sicca.

Rhinitis sicca is not encountered until that time in the life of the individual when general atrophy sets in. It produces no serious symptoms in the nose al

though it is often the source of fairly annoying pharyngitis or laryngitis. The drying, however, may cause the patient to sniff until it becomes an unconscious habit. It is not unusual to see old people who sniff several times a minute without being aware of it, interrupting even their own conversations. If the malnutrition is pronounced, trophic ulcers may occur. These are usually found in locations in which the inspissated secretions collect and produce trauma either by drying and contracting or through the efforts of the patient to remove them. If such ulcers occur on both sides of the cartilaginous septum, perforation of the septum may result.

Where sclerosis of the vessel walls exists there is always danger of epistaxis, which may be severe. It may be spontaneous or it may result from the accumulation of secretions which dry and contract, pulling upon the epithelium and tearing the superficial vessels. While the epistaxis of youth is commonly confined to the regions of the septum immediately adjacent to the nares, the break in a sclerotic vessel more often occurs farther back in the nose and especially at the margin of a sharp septal ridge, a spur or a perforation. Here the vessels are bent sharply across the bony edges which renders them especially liable to injury.

With the deterioration of the special senses come the very distressing symptoms *anosmia* and *parosmia*. These may be the result of persistent infections in early life, during which the olfactory nerve ends are injured or destroyed, or they may be part of a senile atrophy similar to that encountered in the cochlea. They are often interpreted by the patient in terms of taste, as well as smell.

While an anosmia, partial or complete, may be unpleasant by rendering the diet monotonous and unattractive, the parosmias may assume tragic proportions. The patient is pursued by a specific odor which it is difficult for him to regard as subjective. The odor is rarely pleasant but is often described as the smell of burning feathers, of ammonia, or of decay. At the same time he imagines that he tastes something abnormal and attributes the whole syndrome to a foul postnasal discharge or a dental caries which does not exist. The wearing of artificial dentures sometimes heightens the effect.

Sinus Disease. There are no diseases of the sinuses peculiar to old age. It may be said that on the whole infectious sinus disease has a tendency to improve with age, owing partly to fibrosis and partly to improved ventilation brought about by atrophy.

THE PHARYNX

The general muscular atrophy which occurs in old age involves the constrictors of the pharynx and the palate and results in a thinning of the faucial pillars and walls. This atrophy is found also in the intrinsic and the extrinsic muscles of the larynx, as has been recently demonstrated. It affects the voice in very definite ways. First, the walls of the pharynx become more firm and

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But the tonsils in which there have been repeated infections are traversed by a network of fibrous tissue which may prevent the crypts from emptying. Fibrosis of the tonsil begins in the proliferation of fibroblasts surrounding the blood vessels in the trabeculae. This restricts the blood supply to the lymph follicles. These are thus much reduced by the process.

Not only does tonsil atrophy occur in this usual way but, according to Wright, the fibrosis is frequently distributed in such a way that whole clumps of cells are torn away from the tonsil. Islands of lymph tissue become separated from the body of the tonsil by the fibrous tissues contracting. These become pedicles from which the now isolated masses depend and from which they are finally broken off in the act of swallowing. This process of spontaneous amputation of masses of tonsil tissue Wright has termed "autoclasis". During their separation the masses are not unlike papillomata in appearance.

After such extensive atrophy and destruction the crypts are usually wide and shallow, nonretentive and harmless. However, when the network of fibrous tissue preponderates at the mouths of the crypts they are surrounded by tough bands which prevent their emptying and cause them to harbor detritus and pathogenic organisms. The apparent size of the tonsil or of the crypt, bears no relationship to its importance as a carrier and a focus of infection. The small, flat nodular tonsil is more likely to be troublesome than the larger less fibrous one.

The tonsil may therefore be a menace to health at any age and since tonsillectomy can be satisfactorily performed under local anesthesia there is no age limit contraindicating it. Patients in the seventies undergo this operation with relatively little local reaction and often with great relief from arthritis or neuritis.

Atrophy of the lingual tonsil, especially if there is much fibrosis may leave behind tortuous and varicose veins in the base of the tongue which are commonly the source of bleeding following severe coughing in older people. Such bleeding is usually not severe although rarely ligation is required to control it.

Glossodynia, with or without the constant impression of a bitter taste at the back of the tongue is occasionally encountered in middle aged persons. It is apparently a pure parageusia combined with a paresthesia which may be general or may be localized in one or two papillae near the anterior faucial pillar. This condition receives a disproportionate amount of the patient's attention because it is usually associated in his mind with the possibility of malignancy. According to Semon, women at the menopause are particularly subject to hyperesthesia and paresthesia of the pharynx. This may or may not be associated with mental depression. Anemia, gout, alcohol and excessive smoking may produce the condition. It may last several years and subside spontaneously. Parageusia may be caused by the contact of unlike metals in dental fillings which sets up minute electric currents. Unless the cause can be found treatment is apt to be futile.

THE EPIGLOTTIS

The epiglottis in elderly people is apt to be thin and the edge irregular. The color is more often pale than red and there may be tinges of yellow at the margin.

Exudative erythema of the epiglottis has been described as occurring in

women over fifty years of age. It accompanies the rheumatic or gouty diathesis and produces an irritative cough, dysphagia, dry throat, and general malaise. There is a marginal ulceration with slightly thickened edges, but no induration. The epiglottis is characteristically the primary site of the lesion which may spread to the aryepiglottic folds, the palatal structures, and the gums and lips. Xerophthalmia sometimes accompanies the condition. Arsenic over long periods is the recommended treatment. Vitamin A has been recommended also.

THE LARYNX

Certain changes continue to occur normally in the larynx after it is fully developed. Ossification takes place during adult life. Laryngeal ossification is found in males over twenty and in females over twenty-two. The ossification centers appear in the thyroid, the cricoid, and the basal parts of the arytenoid cartilages. The apices and the vocal processes of the arytenoids and the cartilages of Santorini and Wrisberg and of the epiglottis never ossify.

The significance of this ossification to the physician lies chiefly in the obstacle which it offers to surgical laryngofissure. The cartilages, which in younger persons may be cut with heavy scissors, must in later years be sawed. No functional impairment arises from the ossification of the laryngeal cartilages.

Degeneration of the muscles of the larynx has been shown to occur with advancing age. The laryngeal structures become paler and thinner and appear more delicate. These alterations have the effect upon the voice mentioned above. The vocal changes are even more pronounced when they are accompanied by deafness. With the deterioration of the eighth nerve, the patient's voice sounds weak and far away to him, for which he compensates by forcing it. The use of a hearing device may mitigate this to some extent, but only when the high pitch losses are relatively small (see p. 301).

TUMORS

There are no nasal tumors peculiar to old age, although malignancies of the nose occur most frequently between the ages of fifty and seventy and are more often found in men than in women. In the larynx, also, incidence in men greatly exceeds that in women and the time of onset is usually in the sixth decade though it may occur in advanced old age.

Nonmalignant nasal tumors are occasionally reported as occurring in later life. Osteomas and chondromas may occur as late as the sixth decade and chordoma has been found in a man of sixty-eight. Rhabdomyoma, a very rare tumor in the nose, has been described by Cooper in a man of sixty-seven years. Lipoma, rarely found in the epipharynx, was reported by Lawton in that location in a man of seventy-two years.

In older subjects malignant epithelial tumors concern us chiefly. Of these the squamous cell type of epidermoid carcinoma is the most common in the

result of chronic irritation, most often of infection in the sinuses. There may be also an extension of squamous cells from a dental fistula communicating with the maxillary sinuses.

Nonmalignant tumors of the larynx may occur at any age but are most often found in the most vigorous years of life when use and abuse of the voice are greatest. Neurofibromata of the pharynx and larynx are seen in patients of both sexes in late middle life.

Of the malignancies found in the larynx epidermoid carcinoma occurs most frequently. In a series of 380 cases of carcinoma of the larynx Figi and New reported 84 per cent occurring between the ages of forty and seventy, 9 per cent in patients between the ages of seventy and eighty. The proportion between men and women is about ten to one.

CHAPTER 22

CHRONIC DISEASES OF THE LUNG AND PLEURA

J BURNS AMBERSON

ALVEOLAR EMPHYSEMA

THE anatomic and functional changes of the respiratory system which accompany the aging process influence the course of many diseases. The commonest change is alveolar emphysema. Outwardly its most prominent sign is a barrel-shaped chest which assumes a rigid inspiratory position and is associated with atrophy and fatigue of the respiratory muscles. In some cases the muscular and skeletal changes may be aggravated by sclerosis of the intercostal and other arteries, leading to impaired nutrition of the tissues. The conspicuous effect is deficient ventilation of the lungs due to impairment of the bellows action of the chest wall and failure of the lungs, because of diminished elasticity, to recoil during expiration (See Fig 24, p 78). Stagnant gas accumulates in the dilated air sacs. This, together with the reduction of the alveolar surface and the diminished permeability of the alveolar capillary wall, which may be caused in part by fibrosis and vascular changes, interferes with proper oxygenation of the blood and the nutrition of all of the body may suffer because of hypoxemia.

In the bronchi, also, there may be loss of elasticity, interfering with the rhythmic motions which help normally to clear out secretions, foreign particles, and infectious exudates. Hypertrophy, atrophy, and metaplasia of the mucosa may have similar effects. Since the bronchial system serves not only for conduction of air but also for drainage, the importance of these alterations is apparent, they predispose to inflammation and retard its healing.

Practically all aging people develop some degree of pulmonary emphysema, and frequently there are other changes such as pulmonary fibrosis, chronic adhesive pleurisy, and chronic bronchitis. Dyspnea and tachypnea are the most common symptoms and are the mechanisms whereby ventilation of the lungs is maintained as nearly as possible at a normal level. They cause much of the physical limitation and tendency to fatigue which these people experience. If ventilation and gas exchange between the blood and the pulmonary alveoli cannot be maintained adequately, chronic hypoxemia results. This aggravates the symptoms mentioned and may result also in irritability, weakness, anorexia, and impairment of general tone and nutrition.

Treatment. When functional impairment is slight or moderate the dysp-

by supplying extra oxygen by tent or mask for a number of days or weeks un-

til relief is obtained. Often the state may be avoided if the patient appreciates the mechanism and moderates his activities so as to avoid sustained deep breathing. Patients usually can get along satisfactorily even with slight hypoxemia if such simple precautions are taken. Deep breathing exercises are to be avoided but mild exercises may be helpful in preserving the tone of tissues and in maintaining a proper coordination of respiratory muscles. The dietary should be adequate but quite simple and excess weight should be reduced. In some elderly people whose abdominal wall is heavy, atonic, and pendulous, a properly fitted abdominal belt may prove helpful by providing support, raising the diaphragm, and facilitating its respiratory motion.

Climatotherapy So far as known, there is no climate which is of value in directly relieving the functional symptoms of emphysema, in fact, these patients may suffer considerable respiratory embarrassment in high altitudes. A warm mild climate may be helpful to patients indirectly by reducing the tendency to acquire the common cold and helping to shorten its duration. Elderly people usually feel better in warm weather, when they are able to get out of doors for the tonic effect of sunshine and light activity.

Complications Chronic bronchitis with acute exacerbations is one of the most troublesome complications of emphysema and often aggravates dyspnea. Even though the inflammation of the bronchial mucosa is only mild, there may be hypersecretion of mucus and this may be accompanied by tenacious exudate. This clings to the mucosal surface and leads to a partial obstruction of ventilation. Sometimes it causes severe wheezing and asthmatic breathing and there may indeed be an added element of bronchial spasm. The cough may be severe and relatively ineffective, leading to fatigue and exhaustion and consequent impairment of the general condition. Hypoxemia may be aggravated. The several factors involved should be suspected or recognized and treatment should be planned accordingly. Bronchial secretions may be liquefied and induced to flow more freely by the use of steam inhalations and expectorants. The value of most expectorant medicines is questionable but potassium iodide is definitely useful in some cases. Bronchospasm may be treated simultaneously by one of the relaxant drugs given preferably by direct inhalation of the nebulized vapor. The use of one of the antibiotic drugs such as penicillin, given by direct inhalation of the aerosol, is very effective in some of these cases. There are a number of devices to be used for this purpose; recently the inhalation of penicillin dust has been tried. The bactericidal effect of penicillin dust has been demonstrated in the laboratory.

dition may at times be vitally important. For the cough. Some also favor dilute hydrocyanic acid which may be cough mixtures, but as a rule it is better to use a single combination.

PULMONARY FIBROSIS

Etiologic Factors Pulmonary fibrosis is one of the diseases of old age and represents the end result of a variety of causes.

of which is infection. It may be difficult to implicate any prior single severe infection, hence it is logical to assume that a long succession of common respiratory inflammations is responsible.

Bronchopneumonia, which sometimes goes undiagnosed, and especially suppurative bronchopneumonia, may leave their permanent marks (see p 343). The resulting fibrosis usually is of a fine interstitial type and is followed in time by secondary alveolar emphysema. In some cases fibrosis is prominent in the peribronchial tissues, this may involve the bronchiolar wall particularly, obliterating the lumen (bronchiolitis fibrosa obliterans). Associated changes include chronic adhesive pleurisy and chronic bronchitis. As described in the section on Tuberculosis (p 324), the *tubercle bacillus* may be responsible for localized or diffuse fibrosis of the lungs. In elderly people it causes disability chiefly of a functional character, even after the active process has become healed.

Pneumoconiosis, caused usually by the prolonged inhalation of silicious dust in various occupations, leads to disability mainly in the late years of life. At this time—often earlier—tuberculosis may supervene to cause rapid deterioration. After many years the patient with uncomplicated silicosis frequently develops a severe grade of emphysema leading to dyspnea, weakness, and loss of weight. The functional impairment may become so severe that the patient is unable to move about and sometimes he can be sustained only by the continuous inhalation of oxygen.

Fibrosis may result from the inhalation of *irritating fumes and gases*. If the exposure is severe it immediately causes inflammatory edema of the whole respiratory tract, and this may be followed by suppurative bronchitis and pneumonia. The reparative processes then may lead to considerable pulmonary fibrosis, emphysema, and bronchitis.

Radiation fibrosis or pleuropneumonitis formerly was observed principally in women who had received prolonged and heavy radiotherapy for cancer of the breast. Following early exudative infiltration of the underlying lung and pleura, organization takes place, leading to more or less dense fibrosis and shrinkage. The tissues of the chest wall participate. The treated side of the chest becomes rigid, retracted and immobile, and the mediastinal structures and the diaphragm are also retracted. The symptoms include a feeling of constriction, dyspnea, and cough, which is usually nonproductive but occasionally may be associated with mucoid and blood streaked sputum.

Chronic congestion of the lung, caused most often by heart disease, leads in some cases to pulmonary fibrosis (stasis induration) but this is not often of a severe grade.

Sequelae of Fibrosis. Aside from the impairment of respiratory function, pulmonary fibrosis of any variety predisposes to infection. Careful precautions should be taken against colds, and when they occur the patient should remain in bed under medical supervision until all traces have disappeared. Advanced fibrosis often is associated with diminution of the vascular bed of the lungs, and this in turn imposes an increasing burden on the right ventricle of the heart. Compensation is accomplished by hypertrophy of the muscle (cor pulmonale), but in time the characteristic symptoms of right heart failure may appear.

Treatment. The principles of treatment applied in alveolar emphysema should be considered since the two conditions are so often associated.

ASTHMA

Aside from the bronchospasm, which is the principal functional derangement in asthma at any age, the problem in elderly people is complicated by the presence of emphysema. This has been considered already and the special indications for treatment have been mentioned. Obviously the offending allergen, if any, should be eliminated after it is identified. Asthma appears usually to be a nonspecific reaction in elderly people, frequently, therefore, it does not respond to specific elimination or immunizing therapy. Infection is often the "trigger" which sets off the attack. The value of vaccines is questionable and usually nil.

CARCINOMA OF THE LUNG

Incidence. Carcinoma is the common form of malignant disease of the lung and is observed most often after the age of forty. It predominates among men, the ratio in some series being as many as four men to one woman. It is one of the more common forms of cancer, ranging closely in frequency with carcinoma of the stomach. It is observed more frequently in modern times, partly because more people live to the cancer age and partly because of the greater accuracy of diagnosis. It is a question whether there has been an absolute increase in the incidence.

Etiology and Pathogenesis. The cause is no better understood than that of other cancers. Suspected but unproved factors include chronic preexisting inflammatory lesions and the prolonged inhalation of irritating dust, tobacco smoke, and other fumes.

Carcinoma of the lung starts practically always in the bronchial epithelium, and in 70 per cent or more of the cases the original site is in one of the main stem bronchi. The possibility of carcinoma arising in the alveolar epithelium is strongly doubted. The type of cell predominating varies from case to case and also in various parts of the same cancer. Those most commonly observed are epidermoid carcinoma, adenocarcinoma, and undifferentiated types. There is some evidence that the epidermoid type does not metastasize as rapidly as others. When metastases occur they are observed most frequently in the regional bronchopulmonary, tracheobronchial, and mediastinal lymph nodes. Other common sites are the liver and bones. Less often, but still in considerable number, the adrenals, kidneys, brain, pancreas, heart, and pericardium may be involved. The clinical picture varies according to the original site of the growth, its route of invasion, the sites of metastases, and the secondary changes produced in the lungs.

Clinical Onset and Course. Since most carcinomata appear first in a main stem bronchus, the earliest symptoms are usually cough and the production of mucoid sputum which, in many cases, is blood-streaked or occasionally of a currant jelly consistency and appearance. Sooner or later the local irritation of the bronchus may give rise to a troublesome wheeze, noticeable on one side of the chest. At first this may disappear with coughing but later it may persist, usually being more pronounced during expiration. Because the growth usually produces inflammatory changes in the mucosa of the bronchus and encroaches on its lumen, proper drainage and ventilation of the lung is impaired and infection with the common respiratory organisms sooner or later gives rise to varying grades of pneumonia. This may appear as simple bronchopneumonia which fails to clear and may progress into subacute or

protracted suppurative and organizing pneumonia with destruction of the walls of the bronchioles and abscess formation in the parenchyma. The disease runs a constantly or intermittently febrile course, the cough becomes productive of mucopurulent or purulent sputum which frequently becomes foul and copious in quantity. Strength and weight may be lost rapidly.

For some months the clinical course may be that of suppurative pneumonia and the patient may die from this complication before metastases to distant tissues are recognized. Less often infection of the parenchyma is delayed while the mediastinal lymphatics become extensively invaded, producing symptoms such as retrosternal pain, congestion of the neck and head from obstruction of the superior vena cava, and possibly the appearance of a lump in the neck which may be the first superficial manifestation of lymphatic metastases. As the growth invades the pleura and chest wall, persistent boring pain may be a distressing symptom. Pleural invasion may give rise to an effusion of a serous or serosanguineous character.

Carcinomata which start, as a minority do, in one of the smaller bronchioles, may give rise only to limited suppurative pneumonia involving the segment of lung distal to the point of growth. Others which start close to the periphery of the lung may invade the pleura early. If at the apex, a curling syndrome is produced. Invasion of the lower trunks of the brachial plexus causes persistent pain in the shoulder and arm, sometimes with wasting of the muscles. Invasion of the sympathetic nerves or ganglia may cause the classical Hare Horner syndrome, that is, enophthalmus on the affected side, narrowing of the palpebral fissure, and miosis of the pupil. The skin may be flushed on one side of the face, neck, and chest, and it may be dry because of a unilateral anhidrosis.

Metastases may lead to palpable enlargement of the liver, to pathological fracture of a long bone, or to symptoms of a brain tumor. The last, in fact, may cause the first symptom of which the patient is aware. Invasion of the heart or vegetative nervous system may cause various arrhythmias of the heart. Dyspnea may be due to obstruction of the bronchi, pleural effusion, or invasion of the mediastinum. Relatively rarely, metastases appear in the opposite lung.

Diagnosis. The history of symptoms described above, especially in a man above forty years of age, should arouse suspicion. Insidiously developing bronchopneumonia, atypical pneumonia, organizing pneumonia, suppurative indurative pneumonia, or pulmonary abscess in a person of this age is always suggestive and should lead to further investigation. Shoulder pain, if it is not easily explained otherwise, may be the presenting symptom. As mentioned, neurologic symptoms of a brain tumor in an elderly person should always lead to investigation of the lungs as a possible source. Loss of weight and strength, fever, anemia, and anorexia are the principal general symptoms.

Physical examination should be complete, particular attention being given not only to the chest but also to the more usual sites of metastases. Sometimes a small hard node is discovered in the neck just behind the sternum or above the clavicle. The commonest physical signs are dullness with diminution of breath sounds limited to a single lobe or to a whole lung, depending on the site of the growth in the bronchus. Not infrequently high-pitched inspiratory breath sounds are heard together with wheezing rhonchi, loudest near the second interspace or sixth or seventh thoracic spine on one side. The trachea and

heart may be retracted to the affected side because of the organizing pneumonia much less often because of atelectasis. Occasionally obstructive emphysema may be demonstrated in one lung or one portion of a lung. Rales may be scarce although extensive pneumonic changes may be present. Mediastinal invasion may destroy the recurrent laryngeal or the phrenic nerve with resulting paralysis of the vocal cord or diaphragm, respectively.

X ray examination may reveal a homogeneous density of one lobe or an entire lung caused by the pneumonia. If this has not developed, the growth may be visualized in the parenchyma if it is peripheral, otherwise the only manifestation is likely to be enlargement of the hilar lymph nodes on one side with or without widening of the mediastinum.

Bronchoscopy which is always indicated in these cases, is particularly useful because the site of the growth is frequently in the visible bronchi. A specimen taken for histologic examination may then prove the nature of the lesion. Growths in the smaller bronchi may not be visualized, but there may be a suggestive rigidity of the adjacent bronchial wall. Specimens of sputum or of secretions obtained through the bronchoscope, treated as tissue for histologic examination, may reveal the presence of cancer cells. A bronchogram following the introduction of iodized oil into the affected bronchus may demonstrate the point of obstruction but not its nature. When a pleural effusion is present it may show the presence of tumor cells. The demonstration of metastases may be conclusive, particularly if one of these is accessible for biopsy. Surgical exploration of the thorax is indicated in some cases, particularly since surgery offers a prospect of cure if the diagnosis is made sufficiently early. If the growth is peripheral, its nature may be determined only by surgical exploration.

Treatment *Surgical Excision* The only procedure which promises an actual cure of cancer of the lung is surgical resection of the affected organ. In a minority of the cases it may be possible to eliminate the growth by removing only a lobe. Because the main stem bronchus is so often involved as well as for technical reasons the surgeon usually elects to resect the lung together with accessible lymph nodes. After the operation the empty thoracic space may fill with an effusion. In some cases thoracoplasty is performed in order to obliterate the dead space and maintain the mediastinal organs in approximately normal position. The apparent cures are in the neighborhood of 10 per cent of all cases operated upon and found to be operable. There is some indication that epidermoid growths are more amenable, since metastases from these do not seem to occur as early as they do in other types. Earliness of diagnosis

rare and deleterious effects have been observed from time to time. These include principally an aggravation of the suppurative pneumonia and abscess

formation. The use of radium in the treatment of cancer of the lung has been reported by a number of authors. The results have been generally disappointing. The same observation applies generally to the use of radium. Likewise, nitrogen mustard does not seem promising.

Management of Pain and Pressure Symptoms In some cases injection of

the nerve roots or actual neurotomy or cordotomy may be performed in an attempt to relieve the distressing pain. Morphine, even in large doses, is ineffective at this stage. When pleural effusion occurs and persists, as it usually does, evacuation of the fluid may be necessary from time to time in order to relieve symptoms of mechanical pressure.

METASTATIC NEOPLASMS OF THE LUNGS AND ASSOCIATED STRUCTURES

Since all the blood of the body must converge to the lungs for oxygenation it is understandable that these organs are frequently sites of metastases from distant parts. Among the carcinomata which reach the lungs in this way are those involving the stomach, prostate, kidneys, adrenals, and ovaries. Metastases from sarcoma of the bone, teratoma of the testicle, and other types are less common. The lung may be invaded directly by malignant growths starting in the ribs or other contiguous structures. The vehicle for metastases may be either the blood or lymph stream, or, probably most often, both. Usually the metastases are disseminated rather uniformly throughout both lungs, but occasionally, particularly in the case of sarcoma, they may be few and isolated, or even single. In the latter event, the distinction from primary malignancy of the lung may be difficult. Clinically, the patient may give a history of the primary tumor elsewhere, but often enough this is unknown and the only symptoms are those referable to thoracic invasion.

Diagnosis. In the case of disseminated pulmonary metastases the complaint usually is increasing shortness of breath, sometimes with pain and occasionally with slight fever. Loss of weight and strength may be slow or rapid. As the invasion progresses, the dyspnea becomes more severe and the patient may become cyanotic. Cough, which usually is not present at first, may become distressing and productive of only small or moderate quantities of mucoid sputum. It should be emphasized that metastatic malignancy does not usually produce hemoptyses, and, because it does not ordinarily occlude the major bronchi, suppurative pneumonia with its train of symptoms seldom develops. On the other hand, invasion of the pleura is not uncommon, and consequently pleural effusion is observed more often than in primary bronchial carcinoma. Effusion may contribute to the respiratory distress and, on tapping, it may be found bloody but not invariably so. Tumor cells may be found in the fluid. Since the behavior of malignant disease is extremely variable, numerous combinations of symptoms may be found. When invasion of the pleura or chest wall occurs, persistent distressing pain is a common complaint. *The diagnosis depends, of course, on discovering the primary growth, if possible, and on the demonstration of metastases in the chest and possibly at other points as well.*

Physical examination in cases in which the metastases are early and disseminated is usually quite negative except for signs suggesting emphysema and possibly a few fleeting bronchial rales caused by catarrhal reaction in the mucosa. Even as the metastases increase in size the pulmonary signs, particularly

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phangitic types, stringy lines may be seen connecting the nodules. Enlargement of the hilar and mediastinal lymph nodes is most suggestive. Subsequently, the steadily increasing size of the nodules is characteristic.

Diagnosis is more difficult where the metastases are few or single. In some cases the metastases are confined mainly to the mediastinal lymphatics, and occasionally one of these extends through the bronchial wall simulating a primary bronchogenic tumor. Extensive mediastinal invasion may produce the well known symptoms of mediastinal obturation. Bronchoscopy usually is not helpful in metastatic disease, since the main bronchi are seldom invaded. The same may be said of bronchography.

OTHER THORACIC NEOPLASMS

Lymphoma. Next to carcinoma of the lung the most common intrathoracic malignant neoplasms are the lymphomata—lymphosarcoma and Hodgkin's disease. They appear most frequently in the lymphatic structures of the mediastinum, but they may also invade the parenchyma of the lung and the pleura. Parenchymal involvement may occur by direct invasion from affected lymph nodes or, less often, there may be isolated metastatic lesions. Frequently the parenchyma escapes entirely while the growth extends widely through the mediastinum and into the pleura where serous effusion may accumulate. *Leukemic infiltrations* are much less common but these also may present a similar picture within the thorax.

The *clinical picture* of thoracic lymphosarcoma or Hodgkin's disease is the familiar one, together with local symptoms which may be related to mediastinal obturation and pressure, to parenchymal invasion, or to pleural involvement. Pressure or invasion within the mediastinum may cause retrosternal pain, stridulous cough, dyspnea, and wheezing respiration simulating asthma. The veins of the head, neck, and upper extremities may be congested and there may be edema of the chest wall or arm due to lymph stasis. Pleural involvement often results in persistent accumulation of serous fluid which occasionally is associated with a good deal of pain and, more frequently, with symptoms referable to displacement of the mediastinal organs. If the lung is invaded a patient may have persistent cough with the production of small to moderate amounts of mucoid sputum, very rarely a lymphoma will undergo necrosis, break down and discharge through the bronchial tree giving rise to purulent or mucopurulent sputum or even hemoptyses.

The *diagnosis* depends on eliciting the customary history of general symptoms of malignant disease and the local thoracic symptoms described, the demonstration of a mediastinal mass, pleural effusion, or possibly other intrathoracic lesions, and the finding of other evidence such as enlargement of the superficial lymph nodes, of the spleen, and possibly of the liver. The mediastinal growth must be differentiated from carcinoma, tuberculosis, benign neoplasm, and aneurysm of the aorta. Usually this can be accomplished read-

agnostic value in doubtful cases, since lymphomata frequently recede under this treatment while carcinoma remains unchanged. If tuberculosis is suspected, diagnostic radiotherapy should be postponed because of the possible deleterious effect.

Treatment Radiotherapy is the principal means of treatment for malignant lymphoma. Nitrogen mustard is also being tried.

Sarcoma Sarcoma of the lung is very rare. Occasionally, sarcoma or fibrosarcoma appears in the mediastinum or may invade the thorax if it originates in the structures of the thoracic wall.

Benign Tumors Growths of this class arising in the lungs are less common than malignant carcinoma but they sometimes must be differentiated. They include bronchial cysts, chondromas, and adenoma, the latter may give rise to hemoptysis, to secondary infection of the lung behind the growth, and occasionally may invade surrounding structures. Fibroma, neuroma, dermoid cyst, and other less common types comprise the benign tumors of the thorax arising outside the lungs. Since these do not metastasize unless they undergo malignant degeneration, the common clinical manifestations are those due to pressure of the expanding growth. As a rule these tumors increase very slowly in size and attain considerable dimensions before they produce subjective symptoms.

The *clinical picture* then depends largely on the direction of the pressure, which may be on the intrathoracic structures but sometimes also on the chest wall, causing atrophy of the bones and considerable pain. Secondary inflammatory changes in the lung are not often caused by extrinsic pressure on the bronchi and are much less common than in the case of bronchial carcinoma. Occasionally a dermoid cyst may rupture into a bronchus, and the patient may expectorate the contents. The cyst then may become secondarily infected and the sputum purulent and foul. Unless secondary inflammatory changes occur, the patient usually is in good general health. Such benign tumors should be distinguished from malignant ones and in this respect it is helpful to remember that benign growths are usually single, circumscribed, and round or ovoid. They seldom arise in the lungs or bronchi, usually in the mediastinum. Bronchoscopy as a rule is not helpful except to demonstrate bronchial pressure or displacement and to rule out carcinoma. Diagnostic artificial pneumothorax may be useful in proving that the growth does not involve the lung and in localizing the site of origin more definitely. Diagnostic radiotherapy may be helpful.

Treatment depends on the size of the growth, the pressure effects which are being caused, and the age of the patient. Young people and those in their forties or early fifties should have the benefit of the judgment of a competent thoracic surgeon, since there is a prospect that serious damage in later years may result from neglect. Many of these growths have been successfully removed by surgical operation, which is the only cure. However, surgery is rarely to be considered in people of advanced age.

TUBERCULOSIS

One of the most striking features of tuberculosis in aging and aged people is the high and mortal rate of the disease in the aged. The chest is more

serious tuberculosis than any other period of life. Beginning with adolescence there is in almost all communities a sharp rise in the incidence of the disease, usually manifest in its pulmonary form. Among females the death rate rises to its peak in the early twenties, after which it drops to a relatively low level.

during the later years of life. Among males the ascent of the mortality curve continues on through the later years of life, so that among elderly men there is almost always more sickness and death from tuberculosis than among women in the same age groups. This is true both in urban and rural communities. In New York City, for instance, in the five year period 1941-45, the death rate from tuberculosis per 100,000 among white men between the ages of 50 and 69 varied from 114 to 140, whereas that among women was 20 to 37. Similarly in the State of Oklahoma during 1939-41 the relative death rates are of interest, being 22.6 per 100,000 among white men 45 to 64 years of age as compared with 10 among women of similar age. Frequently in these elderly people the lesions are of long duration and chronicity has been established. Many such tuberculous people, who may endure the disease without great disability for years, circulate in communities disseminating the infection among their fellows. *The elderly tuberculous are an important factor in the perpetuation of the disease throughout the world.*

Pathogenesis. Investigations near the end of the last century indicate that in civilized communities tuberculous infection was almost universal and was frequently acquired early in life. This picture has changed materially, particularly in the older and economically favored countries such as the United States, as the incidence of tuberculosis has diminished. Records of the American Student Health Association show that among freshmen entering various colleges of the country, only 10 to 30 per cent react to the tuberculin test. Somewhat similar observations have been made, for instance, in the Scandinavian countries. On the other hand, pathologic studies such as those made by Medlar in New York City, Carnes in Baltimore, and Lande and Wolff in Hagerstown, Maryland, indicate that there is a rapid accretion of tuberculous lesions to be found at autopsy after the age of twenty, rising to an incidence of 70 to 90 per cent in the late decades. This evidence means that many adults now acquire primary tuberculous infection in early adult life or later.

The relative danger of the resulting primary lesions as compared with the exacerbation of old latent lesions is being considered and studied in various clinics. It is known that primary lesions in adult life are not so frequently associated with massive caseous involvement of the regional thoracic lymph nodes or with generalized hematogenous tuberculosis, as is the case in the years of infancy and early childhood. Nevertheless, there is a great potential danger in early active pulmonary lesions in adults whether these are primary or due to the reactivation of preexisting lesions, and the difference is not very striking. If activity of the lesions continues the result in either case is in general the same. Usually the lesions extend, undergo caseous necrosis and ulceration, leading to the discharge of infectious pus into the bronchi. This then is inhaled to greater or less degree and at varying intervals of time into healthy parts of the lung, thus accounting for the variable progression of the disease insidiously or acutely, steadily or intermittently, extensively or in very limited areas. Similarly the infection may reach the surface of the larynx or be swallowed, to come in contact with the mucosa of the intestine leading to the common complications of tuberculosis of the larynx, of the lower ileum, and cecum. Reparative processes are a common feature of the disease, leading sometimes to resolution of inflammatory non-necrotic lesions and to fibrous repair.

Dissemination of the infection through the blood stream is not so frequent in adults as in young infants but it is interesting to note that late hem

atogenous disseminations, sometimes leading to terminal miliary tuberculosis appear to be more frequent in elderly men than in elderly women. Caseous lesions in the prostate are sometimes found to be a source of contamination of the blood stream.

Etiologic Features. Reactions to the tuberculin test indicate that most people harbor at least a few tubercles in their tissues as they pass through adult life, yet relatively few of them ever develop clinical tuberculosis. Hence one must look to causes other than the tubercle bacillus to help explain this. Constitutional and environmental factors undoubtedly play an important role. It has long been observed, for instance, that uncontrolled diabetes mellitus predisposes to the development of pulmonary tuberculosis. Elderly people whose resistance becomes impaired by disease such as cancer, hepatic cirrhosis, and chronic alcoholism may suffer exacerbation of tuberculous lesions which otherwise would probably remain latent. Poorly fed and housed people have a higher incidence of the disease. The factor of sex has been mentioned. In young women the general metabolic and functional effects of the sexual cycle are strongly suspected because of the relatively high death rate at this age period and the striking coincidence of various clinical manifestations with menstruation.

In elderly men particularly, an important influence is found in occupational hazards. Silicosis predisposes to tuberculosis and the death rate from the combination (silicotuberculosis) is relatively high among those working in trades in which there is exposure to silicious dust. Aside from this it is known that poor working conditions, exemplified by unskilled laborers, involve a death rate from tuberculosis which is five to ten times higher than that among professional classes. The factors of low wages and living conditions are of course interwoven. It is self evident that the ultimate conquest of the disease must take into consideration these and other incidental causes as well as the factor of infection alone.

Clinical Onset and Course. *Acute pneumonic tuberculosis* is not encountered as often in elderly people as it is in young adults. When it appears it is usually in a patient whose resistance has been depleted by some complicating element such as uncontrolled diabetes, chronic alcoholism, or malnutrition. The onset and course are not particularly different from those observed in younger people. Occasionally, even in the elderly, the inflammatory lesions may be arrested soon after the onset, followed by more or less resolution of the exudate which has not yet become caseous. The toxic constitutional symptoms may then gradually abate, but usually cavities are left behind in the lung and the disease continues a chronic course. More often, however, the disease progresses as acute caseous pneumonia, and death may occur within six or eight weeks, sometimes preceded by the development of tuberculosis of the larynx and intestines.

Many clinical variations are observed between this and the opposite ex-

long as thirty or forty years. The current exacerbation usually manifests itself
 as a definite loss of weight and strength, dyspnea
 and cough, ex-
 cess occur, con-

sisting frequently of a few streaks of blood in the sputum, fairly often of moderate quantities, that is, up to five or six ounces of blood, and, in a small minority, of massive and sometimes fatal hemorrhages. Streaking of the sputum or small clots may be observed over a period of many weeks. Commonly, pro-

usually shrink in favorable cases and eventually the sputum may disappear or be scant and free of tubercle bacilli. Not infrequently, however, careful culture of the sputum will reveal a few organisms persisting.

Fibroid tuberculosis is fairly common in aging and old people. It consists of lesions which may be predominantly localized in a part or whole of one lung or disseminated throughout both lungs. It is very seldom that one lung escapes completely. The symptoms of fibroid disease are usually milder but more protracted than in the fibroulcerative, partly because of the associated emphysema which is now permanent and irreversible. Dyspnea, especially on exertion, becomes more pronounced. Added to this, distortion of the heart and great vessels, due to fibrosis, may cause circulatory inefficiency with the resulting symptoms of cyanosis, susceptibility to cold, and coldness of the extremities. Chronic anoxia may lead to nervous irritability, impairment of nutrition, and other functional symptoms. The damaged parenchyma and bronchial tubes are more susceptible to the effects of simple respiratory infections, so that associated chronic bronchitis and occasionally bronchopneumonia prove troublesome and serious. Infrequently there are hemoptyses, usually small, which do not originate in excavated areas but from superficial varicosities developing in the bronchi.

Occasionally *acute tuberculous pleurisy* with effusion develops in any one of these forms. This frequently is limited and runs a mild course with eventual absorption, but occasionally the effusion is massive and there are severe febrile and other constitutional symptoms. It may be absorbed, but sometimes becomes partly organized, resulting in fibrothorax with considerable retraction of the adjacent structures.

In some cases tracheobronchial lesions which are secondary to parenchymal foci, prove troublesome and dominate the clinical picture. Tuberculous granulations or ulcers in the bronchi may cause dyspnea, wheezing, which frequently is unilateral, and expectoration of quantities of mucoid sputum. Laryngeal tuberculosis may be severe, causing distressing pain, hoarseness and occasionally dysphagia. The course of the laryngeal lesion often parallels that of the pulmonary, that is, it may be of a mild proliferative type causing only annoying hoarseness and dryness of the throat. Intestinal involvement, centered mostly in the lower ileum and cecum, if ulcerative, occasionally leads to severe colic and diarrhea and serious impairment of nutrition. Much less often a hyperplastic lesion in the cecal region leads to chronic obstruction. A few patients with "open" pulmonary tuberculosis (that is, a cavity discharging bacillus laden pus) develop lesions of the lower end of the intestinal tract, manifest most often as perirectal, perianal, or ischiorectal abscesses and fistulas (see p. 555). These are acutely or subacutely painful and swollen at the start and give rise to a chronic discharge when the fistula is established.

As mentioned above, dissemination of the infection through the blood

stream is not so frequent in the late years of life as it is in infancy, but it is not rare. At the autopsy of elderly subjects who died from pulmonary tuberculosis isolated tubercles have been found in the liver, spleen, and kidneys in 20 per cent or more of the cases as a rule these were not clinically manifest. On the other hand it is important to appreciate that in elderly people vague states of progressive debility associated with intermittent or continuous low grade fever may be due to subacute generalized hematogenous tuberculosis which does not become clearly manifest for weeks or months. Likewise, the peculiar pattern of tuberculosis and its frequently long chronicity explains why isolated lesions in the kidneys, lymph nodes, bones and joints, and other structures outside the lungs may become active, progressive, and destructive in late life even though the infection may have been implanted during hematogenous dissemination of the infection early in childhood.

Diagnosis Nothing is more important than a searching *history*. Familiarity with the intermittent course and the pathogenesis of the disease suggests the questions which are to be asked. The history should start with an inquiry about the possibility of contact, particularly in the family. This may have occurred in infancy or early childhood. Any episode of respiratory illness in the patient's past should be investigated in detail, and the possible relation of extra pulmonary manifestations should not be neglected. The occurrence of lymph adenopathy in childhood, of unexplained prolonged febrile attacks, of an obscure arthritis, of pleurisy with effusion (which sometimes is known to the patient only under the name of pneumonia), hemoptysis recently or at some remote time, persistent cough or clearing of the throat, wheezing dyspnea all should be earmarked as to their nature and duration. If the patient has expectorated pus its quantity, character, odor, and persistence should be known. Persistent or chronic hoarseness, fistula in ano, and chronic diarrhea should always arouse suspicion.

Of the general constitutional symptoms, the most important is unexplained loss of weight or inability to improve the nutrition by adequate diet. Malaise, weakness, and lack of staying power, particularly when working are suggestive. Febrile attacks may be known to the patient only if they are severe. Low fever is often unrecognized because the toxemia is of a peculiarly mild character. It is seldom, for instance, that tuberculosis causes severe chills and when these are described, they should lead to the suspicion of some other cause.

Single symptoms in themselves may be common to many diseases but the combination and sequential relations are so characteristic that one is not often misled. Medlar in a recent study of autopsy experience at Bellevue Hospital has shown that fatal pulmonary tuberculosis in young adults was almost always properly diagnosed before death. On the contrary, the diagnosis was often missed among elderly people, partly because the presenting symptoms in the wards were related to some other disease such as heart disease, and partly because of other diagnostic difficulties. Among the elderly, tuberculosis in its chronic form often simulates cancer, chronic lung abscess, bronchiectasis, aspirational pneumonia, and other conditions, and the differentiation is often

may become active and progressive in the course of some other fatal illness. The problem is important not only for the individual patient and successful treatment, but also for the protection of his associates against infection.

Physical Examination A complete examination of the whole body should be made, otherwise extrapulmonary lesions may be overlooked and these may be of great importance. In examining the chest, inspection and palpation often are of greater help in elderly people than in the young because of the effects of fibrosis. Localized or unilateral retractions of the chest wall and restrictions of its motion may be significant. In addition, the trachea and heart may be displaced. On the other hand, particularly in fibroid disease with emphysema, the chest may be barrel-shaped and rather rigid. The distended, inelastic, and poorly ventilated parts of the lung may obscure signs of tuberculous pathology which otherwise could be elicited. In fibroulcerative types, classical signs of cavitation are sometimes discovered, but the failure to elicit them does not eliminate the possibility of excavation. If the cavity wall is thin and flaccid, the cavity acts as a poor resonator, no striking alteration of breath or voice sounds is demonstrated, and the only significant sign is medium rales. Pneumonic consolidations of part of a lobe, or all of it, usually produce definite signs. The point to remember in differential diagnosis of tuberculosis from pneumococcal pneumonia is that the former usually represents an extension from preexisting cavernous areas in the upper part of the lung but the signs of the cavity may be obscured by emphysema.

X-ray Examination This should be a part of the study of every case, since physical examination alone is inadequate. The appearance of the roentgenogram characteristically is that of a more or less chronic lesion in the upper part of the chest, usually predominating on one side but frequently rather symmetrically distributed in both lungs. Retractions of the thoracic contents and adhesions to the diaphragm may be readily visualized. The signs of emphysema may be striking and the x ray may be of particular value here in revealing tubercles which could not be detected by physical examination. The dense and nodular character of many of the lesions, and the mottled appearance of the

genogram alone may be deceptive and misleading, emphasizing the necessity for complete studies and their proper correlation.

Laboratory Examinations The most important of these is for the detection of tubercle bacilli in the sputum. The patient must be carefully instructed to expectorate and collect the mucopurulent particles or masses which he raises in the morning. Except when the disease is thoroughly healed, fibroid, and devoid of ulcerating areas, the organism should be demonstrated in the sputum. Too much emphasis cannot be placed on the necessity of careful technique, not only in collecting the specimen, but also in making the examinations. If single smears do not reveal the acid-fast organisms, repeated specimens should be obtained and examined during a period of weeks or months, using the more refined methods of sputum concentration, culture and guinea pig inoculation. Failure to demonstrate the organisms is one of the most important ways of disproving the tuberculous nature of a chronic pulmonary lesion.

It should be remembered that fibroid cavities being relatively clean, do

not cast out as many bacilli as freshly caseated ones, consequently, it is often more difficult to demonstrate the organisms in the sputum of an elderly person

Management The necessity for careful treatment of obviously progressive or pneumonic forms of tuberculosis is self evident. Among the many fibroid cases, however, the indications for immediate treatment are not always clear, and careful judgment must be employed in order not to inconvenience the patient unnecessarily, but still to avoid possible progression of the disease when it might be easily arrested by prompt action. These fibroid lesions are frequently limited to the upper part of one or both lungs, and as a part of the diagnosis it is necessary to determine their actual or potential threat to the patient's health. Lesions confined to a small area, such as that above the first or second rib, which appear fibrotic and calcified and produce no symptoms, seldom require active treatment in elderly persons. Such people should have routine examinations *including roentgenograms at least once a year* in order to make sure this assumption is correct. Lesions which are more extensive and less definitely fibroid require a period of observation for further manifestations. X-ray examination every two to four weeks at first may reveal progressive or regressive changes, either of which indicate a potential threat. Aside from this, the appearance of any symptoms of toxemia or of cough with expectoration, the finding of tubercle bacilli in the sputum, or the finding of an accelerated sedimentation rate of the red blood cells or of an abnormally high leukocyte count, are of similar significance. Tuberculosis may be slowly progressive, however, without changing the sedimentation rate or the leukocyte count materially. In such cases, when there are no signs of instability of the lesion, periodic observation is all that is necessary, provided the patient is advised to follow a careful, hygienic routine of living.

The subacute or indolent types of fibrocaceous or fibroulcerative tuberculosis, as well as the pneumonic types, require long periods of rest in bed, until all the symptoms and other evidence of toxemia have subsided and until the serial roentgenograms indicate stabilization of the lesions. The patient should be allowed little or no activity for three to six months after the lesions

appearance of tubercle bacilli from the sputum. The healing powers of elderly people are not to be underestimated, although the necessity of prolonged rest treatment should be understood (See p 32.) The possible disadvantages of immobilization of these people, and such risks as thrombosis of the leg veins and pulmonary embolism have to be considered. Since tuberculosis in the elderly so frequently runs an indolent course it is usually permissible for the patient to have lavatory privileges, seeking to avoid such disadvantages. Passive exercise of the limbs and massage also help to promote circulation and preserve tone of the tissues.

In arranging the dietary the desirability of maintaining good alimentary function and of supplying necessary vitamins, minerals and other elements is the guiding principle (See p 193.) There is no virtue in an excessive gain in

as we know, there is no specific value in any element of the diet, it should be designed to maintain metabolism at a normal optimum. When complicating tuberculosis of the intestine exists it is necessary to modify the diet, which should be bland and as free as possible from indigestible fiber. These patients may not tolerate much fat or fresh fruit so that vitamins will have to be supplied for a time by parenteral injection. The intestinal lesions may tend to improve, even to heal, as the general resistance improves.

The question of a change of climate often arises, and this requires individual consideration. Elderly patients do not profit by cold mountain climates, particularly if the circulation of blood is impaired. Those who have fibroid tuberculosis with considerable emphysema may not be able to tolerate high altitudes where the rarefied atmosphere may increase the burden of the heart and actually lead to failure. As a rule these people do better in mild balmy climates where barometric changes and variations in humidity are not extreme or rapid. Other considerations, however, may necessitate a compromise and

the patient may be sent to a sanatorium where the climate is more favorable. The climate in which this routine is carried out, and some patients do better at home because their accustomed environment entails less strain and readjustment. Generally speaking, however, a properly managed sanatorium in which the medical and nursing routine is carried out by skilled professional people is the best, particularly at the start. Many of these patients require a year or two of treatment before the disease can be brought to arrest and it is important that the start of the treatment should be under good auspices. Eventually a considerable number arrive at a point where the disease becomes quiescent, although fibroid cavities remain, giving rise to cough and expectoration of sputum containing tubercle bacilli. Living in comfort under sheltered conditions, such people may survive for many years. The intelligent will voluntarily use precautions to avoid the infection of healthy people, but the ignorant and intractable are a menace in many of our communities and at times they may have to be forcibly segregated.

Streptomycin, discovered by Waksman and his associates, is the first chemotherapeutic agent found to have a definite inhibitory effect in clinical tuberculosis. This antibiotic has the disadvantage when used in effective doses of frequently causing damage to the vestibular apparatus, which damage appears to be permanent. Furthermore, the tubercle bacillus may become resistant to the drug after a period of two or three months or even less. On the positive side, administration of the drug in acute generalized miliary tuberculosis and tuberculous meningitis, which almost always are otherwise fatal, frequently leads to an arrest of the disease, in a percentage of these cases, especially those with meningitis, a fatal relapse occurs after some weeks or months. In the pulmonary form favorable effects are noted in many cases of recent acute tuberculous pneumonia (especially if not more than two to four weeks old). In the more chronic phases the effects are less striking and may be equivocal or nil. Benefit has been observed in tuberculosis of the larynx, bronchi, intestine, and in superficial lesions associated with draining sinuses. The scope and limitations of streptomycin are being widely studied but have not yet been well defined. When he uses it the physician should proceed on the as-

sumption that residual tuberculous lesions will persist, requiring rest treatment or other management, and should avoid giving the patient a false sense of security because of early favorable response to the treatment. Furthermore, he should not underestimate the disadvantages mentioned above.

Collapse of the lung by artificial means, designed particularly to close pulmonary cavities and prevent further progression of the disease, is sometimes a distinct aid even in those who are in the fifth decade of life. Occasionally, artificial pneumothorax may be sufficient to bring about the desired result when rest alone fails. It must be remembered, however, that pleural adhesions which are associated with chronic disease often interfere, and the inelasticity of an emphysematous lung may prevent as prompt a collapse of the lung as might occur in younger people. Dyspnea is more likely to be a symptom in the elderly and this impairment of function may actually preclude the use of such therapy. *Surgical operations*, such as thoracoplasty, extrapleural pneumothorax and the drainage of cavities by the Monaldi technic, may be considered in aging people when proper indications are present, when the general condition is good, and functional impairment is not very great (See p. 143.) Results, however, are not so encouraging as might be expected in younger adults.

SUPPURATIVE PNEUMONIA AND PULMONARY ABSCESS

Etiology. Suppuration is a factor of many bacterial pneumonias but under certain conditions some of the pyogenic bacteria may cause acute and severe suppurative inflammation with necrosis of the lung leading to abscess formation. The process may be wide and diffuse but more frequently it is confined to one lung where the suppurative pneumonia is associated with a single or multilocular abscess. Occasionally there may be predisposing factors such as thrombosis of the pulmonary vessels, infarction, invasion of the lung by infection in the pleura, interference with bronchial drainage by a bronchial tumor or foreign body, or damage of the lung by inhaled irritating gases or other substances. In some cases the suppurative pneumonia is of a so-called nonputrid character, in which case it is usually due to a single pyogenic invader such as staphylococcus or Friedlander bacillus. Putrid suppurative pneumonia, identified clinically by the odor of the breath and sputum, is almost always associated with anaerobic organisms indigenous to the mouth and nasopharynx. The pneumococcus seldom is responsible for lung abscess. Infection occurs by one of two main routes, namely, from septic emboli which lodge in a pulmonary vessel, or by inhalation or aspiration from the mouth and upper respiratory tract.

The latter is much the more common mechanism in elderly people. They have more carious teeth and more pyorrhea alveolaris, which is a common source of aspirational infection. Their protective pharyngeal and laryngeal reflexes are often impaired as age advances so that aspiration is easier. Changes in the bronchial structure, particularly the mucous membrane, may have occurred gradually through the years, so that ciliary action does not seem to be as effective as in younger people. Finally, the emphysema of advancing age, which is an easier infection route, could be dis-

explain the less satisfactory response to treatment.

Suppurative processes following the lodging of septic emboli are fre-

quently due to infection with a single organism such as a staphylococcus, the nidus of which may be in some distant focus such as osteomyelitis

Diagnosis. Following the familiar onset, which cannot be distinguished at inception from that of simple bronchopneumonia, the clinical course is characterized by prolongation beyond the expected time and the symptoms of suppuration, i. e., the expectoration of quantities of purulent sputum which, in the case of an anaerobic infection, soon takes on a fetid odor and may become very copious. Unless treatment is effective within the early weeks, the destructive process is likely to spread, involving wider areas in one or both lungs, or it may settle down to a chronic abscess with organized walls. In the latter event, healing in the strict sense of the word may never take place, cavitory defects remain in the lung with the possibility that recrudescence of inflammation may occur at any time.

Since early diagnosis is of vital importance in the prognosis of these cases, careful attention should be paid to any attack of pneumonia which does not follow the expected course. Other points of importance are the appearance of purulent expectoration which increases in amount and continues even after the fever subsides. Foul sputum should always arouse suspicion. X-ray examination is indicated for all patients who have had pneumonia in order to determine whether any residue remains after apparent clinical recovery, particularly when the disease has run an atypical course. The sputum should be examined bacteriologically to identify, if possible, the causative organisms, it is always important to search for tubercle bacilli.

Treatment. The pulmonary lesion should be accurately localized by suitable x-ray examination. Diagnostic bronchoscopy is indicated, it serves to determine a possible local cause, such as a bronchial carcinoma or a foreign body. Strict bed rest is enforced during the active stages of the disease and for several months afterwards. Since adequate drainage is the most important condition of healing, this should be promoted by enforcing postural drainage for periods of ten minutes to one-half hour several times a day. When the patient is too feeble for this, it may help to raise the foot of the bed and place him in a position to favor drainage of the pus from the involved area.

Chemotherapy has a place in the treatment of suppurative pneumonia with abscess but its limitations should be appreciated, recognizing that inadequacy of drainage of the abscess through the bronchi may prevent a favorable effect. Abscesses due to single aerobic pyogenic gram-positive organisms, such as staphylococcus or streptococcus, have a great tendency to heal spontaneously and this is accelerated by the administration of penicillin or sulfadiazine. Chronic infection with the Friedländer bacillus does not appear to be influenced by these drugs but streptomycin is being tried with some reportedly favorable influence, on the other hand the bacillus may become drug-fast in a few days. In putrid lung abscesses due to anaerobes the effect of chemotherapy is equivocal in most cases. After a short trial of parenteral injection of penicillin, if there is no favorable response, it is usually wise not to rely too heavily on this means of treatment. It is doubtful whether in the acute phase there is any advantage in the administration of the drugs in aerosols.

In any event the course of the lesion should be followed very closely by the temperature curve, a record of the daily amount and character of the sputum, and roentgenographic examination of the chest every week or ten days during the early phases. Satisfactory response in cases in which the di-

agnosis is made reasonably early is indicated by defervescence, diminution of the sputum which loses any foul odor it may have had, becomes mucopurulent, then mucoid, and finally disappears, and x-ray evidence of resolution of the inflammation and contraction and obliteration of cavities. Physical examination is less helpful except as it may confirm other findings. If the outcome is satisfactory, healing should take place within four to eight weeks. Rest treatment should be continued for two to six months after the lesion has apparently healed, otherwise recrudescence of the latent infection may occur. Complete convalescence with a gradual return to active life often requires six months to a year. If recovery has not taken place in the time mentioned, and the suppurative process has become somewhat localized with formation of a single or multilocular abscess, surgical drainage with thoracotomy is usually indicated. In such cases the opinion of an experienced thoracic surgeon should be obtained. The temporary improvement of symptoms in this situation is very deceptive. There is a temptation to postpone surgical intervention, and this frequently allows the patient to drift into chronic disease from which he never recovers. It must be remembered that chronicity of an abscess may become established within several months.

Since most of the severest abscesses in elderly people are aspirational in origin, the best preventive is to clean up septic foci in the mouth and upper respiratory tract. Putrid pulmonary abscess is seldom observed in edentulous people.

ASPIRATIONAL, CHRONIC, UNRESOLVED, AND LIPID PNEUMONIAS

Pulmonary lesions of these varieties are observed more frequently in elderly people than in the young. Usually careful investigation will reveal a mechanism which explains the peculiar behavior of the disease. The clinician should never be satisfied, without thorough search, to discharge a patient with a diagnosis of unresolved or organizing pneumonia, since the underlying mechanism may be of serious significance and its discovery may be of great importance for the patient's future.

For reasons which have been described, aspiration of infectious discharges from the mouth and upper respiratory tract occurs fairly frequently in elderly people, and if this is prolonged or repeated, the result may be chronic inflammation of the lung which goes on to suppuration and organization—*aspirational pneumonia*. Recognition of the mechanism and proper prophylaxis may prevent chronic ill health.

Abnormalities which frequently result in severe pulmonary damage include repeated regurgitation and aspiration of stagnant esophageal contents in cases of cardiospasm with secondary esophagitis and in cases of diverticulum of the esophagus, especially the former (see p. 513). As the protective pharyngeal and laryngeal reflexes become dulled, the regurgitated material is partly aspirated into the respiratory tract, especially during sleep. Sometimes this results in acute lung abscess, but more often in *chronic suppurative and organizing pneumonia*. This may be bilateral and extensive and eventually may be confused with carcinoma. It may also result from leakage of gastric contents through a fistula due to

trauma, malignant disease, or a congenital defect.

Another form of aspirational pneumonia is *lipid pneumonia*, due some-

times to the aspiration of oil used habitually for the relief of nasal symptoms. The protective reflexes usually are diminished and the oil dropped or sprayed into the nose is aspirated, leading to slowly developing, chronic organizing pneumonia. A similar process, even more extensive, is observed in patients with neurologic or psychic abnormalities who habitually take mineral oil for the relief of constipation. The organizing pneumonia leads to progressive limitations of the functional capacity and predisposes to infectious suppurative pneumonia.

Other causes of atypical, chronic, or organizing pneumonia include *bronchiectasis*. In any prolonged atypical pneumonia such an underlying mechanism should be considered. It is also observed that pneumonia runs a more prolonged and atypical course in *emphysematous* people, presumably because drainage of the lung is hindered and the vascular bed is reduced. Roentgenographically, the lesions are more patchy and chronic looking than in the case of an otherwise healthy person.

Another common cause of chronic or atypical pneumonia is local *bronchial irritation or obstruction*. After the age of forty, bronchial carcinoma is always a consideration, and the finding of such pneumonia may be the principal clue to the diagnosis. Nonmalignant lesions include bronchial adenoma or granuloma, strictures due to old inflammatory processes, and, occasionally, ulcerations and obstructions caused by the perforation of a calcified lymph node into the tracheobronchial tree. An aspirated foreign body may have the same effect.

The development of chronic or unresolved pneumonia is favored, of course, by congestion and edema of the lungs from any cause, the commonest of which is cardiac disease. A wet lung is susceptible to infection by organisms which, in the healthy person, might gain access to the respiratory tract but would be quickly disposed of. Edema fluid or any other fluid in the alveoli or the bronchial tree is often a good culture medium, accounting for mild pneumonia which persists and tends to organize.

MYCOTIC INFECTIONS OF THE LUNG

Mycotic infections are observed somewhat more frequently in elderly people than in the young. *Actinomycosis* is the most common form in most geographic areas, especially rural, in the West and Southwest, *coccidioides* infection is more prevalent. The prevalence of *histoplasmosis* in the central sections of the United States is under study; the identification of the disease is difficult and the epidemiology is still uncertain. At the onset these infections may be of a subacute or even acute pneumonic character, but later they may

demonstrate that the organisms actually caused the lesions, the possibility of confusion is somewhat greater in elderly people because of purulent foci in the mouth and upper respiratory tract which may harbor organisms of this group. In most communities fungus infection is very uncommon, and disease as-

cribed to it is usually found to be of other etiology. *Monilia* and *aspergillus* are found in the mouths of a good many people and they may become secondary invaders of other lesions in the lower respiratory tract without actually contributing to the pathology otherwise, this is particularly true of cavitory lesions.

OTHER GRANULOMATOUS LESIONS OF THE LUNGS

In recent years *pulmonary sarcoidosis* has attracted increasing attention because of the frequent x-ray demonstration of the lesions. The etiology of the disease is still unknown, some believe the tubercle bacillus is the cause while others think that the lesions are quite nonspecific. The disease must be considered in a patient who has, in addition to bilateral fibrosis and nodular infiltration of the lungs, generalized lymphadenopathy, splenomegaly, sarcoid lesions of the skin and other lesions, such as uveitis and parotitis. Tuberculosis and carcinomatosis must be differentiated.

Syphilis of the lung has been described as a disseminated granulomatous infiltration with fibrosis. This is rare and I have never observed a proved case. Gummatous infiltration of the trachea and bronchi is described, but it is very uncommon.

BRONCHIECTASIS

Etiology. Pathologic dilatation and destruction of the bronchial wall is usually the result of protracted or repeated bronchopneumonias in childhood. The fragile bronchial structure is more or less destroyed by the necrotic process and while repair may occur, this frequently leaves behind a bronchus permanently impaired in function and structure. This is particularly true if the myoelastic layers are destroyed. The reparative processes include fibrous tissue proliferation in the bronchial wall, development of new blood vessels, and alteration of the epithelium which may become cuboidal or even squamous. Polypoid outgrowths may appear in the bronchial lumen and there may be areas of chronic ulceration. The permanent dilatation of the tube may be slight and cylindrical or it may be wide, irregular, and saccular. The small peripheral bronchioles are most frequently affected. There is a variable degree of involvement of the adjacent pulmonary parenchyma and of the pleura manifested by fibrosis, emphysema, chronic pneumonia, and fibrous pleurisy. When the chronic pneumonia has been extensive, organization may result in shrinkage and dense induration of the whole lobe or even of the lung. The lesions are most frequently unilateral.

Due to reparative processes which may be fairly effective in childhood, the inflammatory process may subside and for many years the patient may be free of symptoms except for a little bronchorrhea, especially after acute colds. Then subsequent respiratory infection may lead to reinfection of the damaged areas or exacerbations of the old process. Finally, particularly in adult life, anaerobic infection may develop from organisms which normally reside in the

purative bronchitis and bronchopneumonia. Those who survive are often plagued with ill health, at least with recurrent attacks of bronchopneumonia. Most of them sooner or later develop chronic cough, productive of mucopurulent or purulent sputum which may become foul and copious. Hemoptyses are

frequent and, while seldom fatal, they may be large and may be followed by pneumonia. Chronic infection leads to increasing pulmonary fibrosis and the secondary emphysema accounts for the development of permanent functional impairment manifested by dyspnea, wheezing, and possibly cyanosis. Most of these sufferers become totally disabled, socially ostracized, and miserable, and most of them have their lives shortened by this disease. Death is usually from an attack of pneumonia but other complications may enter, such as empyema and metastatic brain abscess. The latter, however, is not so common as sometimes suspected.

Diagnosis The diagnosis depends first on a careful *history* which should include information about the respiratory system from birth. Early attacks of bronchopneumonia, measles, and whooping cough may be significant. Protracted winter coughs, repeated bronchopneumonias, chronic bronchitis, occasional hemoptyses or streaking of the sputum, febrile attacks with mucopurulent, purulent, or sometimes foul expectoration, all help to make up the clinical picture.

On *physical examination* the most important finding is persistent rales at the base of one or sometimes both lungs, usually posteriorly. It should not be forgotten, however, that bronchiectatic areas may be small and confined to a part of one lobe, upper lobe involvement is not rare. Aside from rales, signs depend on how much pneumonia, fibrosis, and organizing pleurisy is present. A cornified lobe may give signs confined to a triangular area along the spine at the base of the lung where dullness, bronchial breathing, and other signs of consolidation are elicited. The signs of upper lobe bronchiectasis simulate those of fibroid or fibroulcerative tuberculosis.

The *roentgenogram* of the chest shows most often a stringy fibrosis following the normal vascular routes in the area involved. During acute phases, however, it may reveal the picture of bronchopneumonia, and, if the disease is of long standing, fibrosis may be diffuse and dense with associated signs of shrinkage and even displacement of the mediastinal structures.

The failure after exhaustive examination to demonstrate tubercle bacilli in the mucopurulent or purulent *sputum* is most significant. The sputum should be measured and observed for its quantity and for the characteristic layering upon standing.

The *bronchogram*, that is, a roentgenogram taken immediately after the instillation of iodized vegetable oil into the diseased area, provides a vivid demonstration of dilated bronchi.

Treatment While the results of lobectomy performed in young people are most promising of cure, the possibilities of this operation should not be overlooked in those of middle age. In well selected cases the operative mortality is below 5 per cent in the experience of competent thoracic surgeons. When the disease is fairly well localized in one lobe, and emphysema and functional impairment have not developed too far, lobectomy is the treatment of choice. Occasionally more than one lobe may be removed, and, in young people particularly, bilateral lobectomies have been successful.

When operation is contraindicated, the elderly patient with bronchiectasis should be advised to follow a carefully planned hygienic routine. He should guard against acute respiratory infection and, if he can afford it, he may spend his winters in a warm mild climate. The mouth should be kept clean, and pyorrhea should be eliminated—if necessary, by dental extractions. Purulent foci

in the upper respiratory tract should be treated. Postural drainage is the best means of removing stagnant mucus or mucopus from the damaged bronchi; this should be practiced once to three times a day, even if the yield of exudate is small. Bronchoscopy may be indicated to promote drainage. Hemoptysis and bouts of pneumonia must be treated with rest in bed, and convalescence may require several months before active infection subsides. Chemotherapy may be valuable for the control of infection in the bronchiectatic tubes and temporary alleviation of symptoms. The bacterial flora should be identified and penicillin or sulfadiazine may be tried to control infection with gram-positive pyogenic organisms. This may be given parenterally or in an inhaled aerosol. Some success has been obtained also by administering streptomycin to control the gram-negative infection. Other drugs are of doubtful value. Injections of iodized oil into the bronchi as a treatment is to be condemned. In my experience x-ray therapy has been disappointing. The general resistance should be maintained, as in any other chronic infectious disease, by rest and proper nutrition.

CYSTIC DISEASE AND BULLOUS EMPHYSEMA

Much confusion has surrounded these conditions, particularly with reference to their origin. The assumption is that many of them represent congenital defects. On the contrary, evidence accumulates that the changes, demonstrated best in the living by x-ray examination, are principally the result of previous inflammatory disease.

Etiology. *Bullous emphysema* may appear as large cyst-like gas-filled blebs, and clinically the symptoms usually are those of simple emphysema. The bullae in some cases develop as a result of repeated bronchopneumonia or other forms of respiratory infection, and often they are associated with pulmonary fibrosis. A simple pulmonary abscess may undergo healing, leaving behind a cyst-like defect which becomes lined with epithelium growing in from the bronchial mucosa. Certain forms of bronchiolectasis present as cyst-like dilatations of the terminal bronchi, and at times this too is associated with bullous emphysema. True cysts of congenital origin are rare and, when present, may appear as gas- or fluid-filled spaces.

Treatment. The treatment of the condition, which is most frequent in elderly people, depends first on obtaining a clear conception of the origin and nature of the lesions. The management then is the same as that for simple emphysema so far as functional symptoms are concerned. Since repeated and protracted infection, which may become suppurative, is one of the main hazards, special precautions should be taken to avoid acute colds and to allow for a sufficient period of convalescence when they occur. Climatic treatment is desirable in some cases. Surgical removal of the cyst-bearing lobe may be indicated if the patient's condition warrants and symptoms are severe. In still others, the cyst, if infected, may require drainage by thoracotomy, that is, it is treated much the same as any pulmonary abscess.

DISEASES OF THE PLEURA

Effusions. Serous and serosanguineous effusions in elderly people always

ally are traceable to an old tuberculous or nontuberculous pleurisy or empyema which has remained in the pleura for a number of years. It may also be caused by hemothorax which has organized and led to chronic changes.

Empyema. Chronic empyema, which frequently is sacculated, may remain latent for many years without producing obvious symptoms. There is a hazard of reactivation of the process or of reinfection, and in some cases, after a lapse of twenty or thirty years, the fluid collection may perforate the visceral pleura and discharge through the bronchial tree. If the empyema is tuberculous, this may result in acute fatal tuberculous pneumonia since tubercle bacilli may remain viable in the pleura for a long period of time. The pleura may become calcified because of the empyema, serous pleurisy or hemothorax, and sometimes, in the case of sacculated empyema, a completely calcified shell is formed.

Pneumothorax. In elderly people, pneumothorax is not so frequently due to tuberculous ulceration of the pleura as it is in young adults. The atrophic changes associated with pulmonary fibrosis and emphysema sometimes lead to thinning of the visceral pleura and the production of subpleural blebs in which gas accumulates under tension. Rupture of one of these is a common cause of pneumothorax. If the emphysema is extensive and function has previously been severely compromised, the pneumothorax may cause death suddenly or within a short time, and the clinical picture may be confused with that of fatal heart disease. Prompt treatment to evacuate gas from the pleural cavity, thus relieving the tension, may be a life saving measure. The pleural tear then may heal quickly and the lung may reexpand within a few weeks or several months. Occasionally it remains collapsed and the tear may have to be repaired surgically. Oxygen inhalation therapy is of great value in these cases. When the pleura tears or an adhesion parts, a blood vessel may be opened leading to hemopneumothorax. The patient then may become exsanguinated. The condition must be recognized and treated promptly if a serious outcome is to be avoided.

Neoplasms. Primary malignant tumors of the pleura are rare. A mesothelioma may originate in the pleura and extend widely, usually manifesting itself by a chronic recurrent effusion which may be serous but, also, sometimes sanguineous. In these cases it is often a difficult problem to make a diagnosis since a peripheral bronchial carcinoma may cause similar manifestations. It is well to remember that pleural effusions due to malignant disease are often related to metastases arising from the stomach, prostate, or some other distant focus. Hodgkin's disease and lymphosarcoma usually arise in the lymphatic structures of the mediastinum but there may be invasion of the pleura with effusion.



CHAPTER 23

ACUTE DISEASES OF THE LUNGS AND PLEURA

HOBART A. REIMANN

INTRODUCTION

PNEUMONIA has long been regarded as a friend of the aged because it so frequently causes peaceful death, either as a primary disease or as the terminal stage of some other acute or chronic malady. It is often called senile, asthenic, hypostatic, or terminal pneumonia. For a number of reasons, the course of the disease in the aged often does not follow the acute violent form observed in younger patients. It is probable that many changes, both general and locally in the lungs, which come with advancing years account for the differences. On the other hand, any of the numerous forms of pneumonia may

so often found in the aged, such as arteriosclerosis, degenerative cardiorenal-vascular conditions, neoplasms, or chronic pulmonary disease, further tends to reduce that state called resistance. It is agreed that resistance or immunity in general, and against the pneumococcus in particular, is a relative and labile state which varies with age. It is low in infancy, increases in childhood, reaches its height in adult life, and diminishes in old age. Measurements of the pneumococidal power of the blood at various ages substantiate this view. Exceptions, of course, exist. The ability to develop specific agglutinins and hypersensitivity or allergy for the pneumococcus varies in a similar manner and serves as a partial explanation for the difference in the behavior of pneumonia in different periods of life.¹ The general state of resistance is probably related directly to the reactivity of the central nervous system which also functions at a progressively lower degree of efficiency as age advances.²

Local Changes. The reflexes in the mucosa of the respiratory tract in advanced age are less sensitive and less reactive. Together with diminished activity of the cilia as a result of senile atrophy or from repeated or continuous low grade inflammation (bronchitis, etc.), this means that a less effective barrier is raised against the passage of pathogenic microorganisms toward the lower portion of the respiratory tract than is found in youth. As a result, microorganisms more easily gain access to the lungs and may incite disease with little provocation. The existence of mild infection of the upper respiratory tract or the presence of chronic inflammatory changes enhances the liability of invasion.

The lungs in old age normally undergo atrophic changes similar to those in the rest of the body (see p. 78). There is a gradual loss of muscle and elastic tissue, often with replacement by fibrous tissue. As a result, the lungs become less expansile and in consequence the weakened respiratory

excursions are less able to assist in expelling mucus, or other secretions laden with potentially invasive pathogens

According to William Snow Miller, the amount of lymphoid tissue increases with age, probably as the result of oft repeated pulmonary infections or of the presence and accumulation of inhaled foreign material, such as carbon or silicon particles (anthracosis or silicosis). The increase of lymphoid tissue is especially noted where the bronchiole divide into the bronchiole respiratori and at the end of the ductus alveolaris. In many cases the amount of foreign matter present may obscure the lymphoid tissue. Anthracosis, except in its most severe form, does not seem to increase the liability to pneumonia, but does tend to interfere with the process of resolution.

The amount of lymphoid tissue and foreign matter in the pleura is also increased in old age, especially in persons who have lived in a smoky or dusty atmosphere.

Pneumonia in General In old persons, as in those younger, it is important to regard pneumonia not as a single disease but as composed of a group of entities, different forms of which have a different prognosis and require different treatment. In the great majority of cases, however, due to circumstances peculiar to old age, the pneumonia is caused by, or associated with, a mixture of the bacteria which are found normally in the respiratory tract. The specific forms of pneumonia caused by single varieties of bacteria or viruses are usually the result of "primary" infection or follow other mild infections or conditions. The chief causes are pneumococci, hemolytic streptococci, staphylococci, Friedlander's bacilli, Pfeiffer's bacilli, influenza virus and other viruses, many of which are not yet fully identified.

As stated previously, pneumonia in the aged may be clinically typical when caused by a single causative bacterial or viral agent, but more often it is not. There may be nothing more than low fever, drowsiness, slight cough, and a few abnormal physical signs in the lungs to indicate its presence. Under these conditions the adjectives broncho, senile, terminal, hypostatic, or asthenic are often applied. Pneumonia may be unsuspected during life.

The incidence and the death rate for lobar pneumonia are highest at both extremes of life. The incidence is lowest between the ages of fifteen and twenty (644 per 100,000) and increases rapidly thereafter, becoming highest after seventy (2000 to 3500 per 100,000). When pneumonia assumes the atypical or bronchopneumonic form, the death rate is higher than in the typical lobar form.

BRONCHOPNEUMONIA, ATYPICAL, SENILE, ASTHENIC, HYPOSTATIC, TERMINAL PNEUMONIA

As just stated, these forms of pneumonia are by far the most common ones in old age. They are often called atypical pneumonia, or bronchopneumonia, to distinguish them from the classical pneumococcal lobar form. In the majority of cases, pneumonia is not recognized clinically and is discovered only if a roentgenogram of the chest is made or at necropsy. It has been said that pneumonia often develops because the person is dying and not that the patient dies from pneumonia.

Pathogenesis and Etiology Because of the multiplicity of factors in old age which lower the resistance to infection, whatever bacteria are already

present in the respiratory tract may gain entry into the lungs and cause pneumonia. Inefficient functioning of the normal mechanism to expel bacteria-laden secretions, infarcts, areas of atelectasis, hypostatic congestion of the lungs, tumors, emphysema, bronchiectasis, and other conditions all favor the development of pneumonia. The presence of other chronic disease such as arteriosclerosis, diabetes, and cardiorenal disorders so common in senility also are predisposing factors, but perhaps most important of all is mild infection of the upper respiratory tract. Repeated attacks of pneumonia are common, if recovery from the first attack occurs.

Because of certain causative factors, still other adjectives are often applied to the pneumonia as follows: (a) *Aspiration pneumonia*, which occurs when the glottal reflex is diminished, thus permitting the passage of bacteria-laden secretions into the lungs during anesthesia, narcosis, coma, or alcoholic intoxication, or in the presence of tumors or debility. (b) *Atelectatic pneumonia*, when areas of atelectasis become infected, this form occurs with chronic bronchitis, bronchiectasis, aspiration pneumonia, and tumors when the excretions are not normally expelled and cause plugging of bronchi or bronchioles and collapse of the related pulmonary tissue. The development of the condition is favored by tightly applied bandages or casts about the chest, and in patients obliged to lie on the back for long periods for various reasons. (c) *Hypostatic pneumonia* commonly occurs in debilitated persons, especially during prolonged recumbency. It develops in areas of the lung where congestion of capillaries, stasis, and transudation of edema fluid into the alveoli occurs, particularly as the result of circulatory failure or in shock or shock-like conditions.

In the majority of cases a mixture of bacteria is found in the involved portions of the lung, usually scattered in the lower lobes. Pneumococci, particularly of the higher numbered types, streptococci of different sorts, staphylococci, Pfeiffer's bacilli, Friedländer bacilli, diphtheroids, colon bacilli, *N. catarrhalis*, and others which are all common inhabitants of the nose and throat are usually present in varying proportions and combinations so that it may be impossible to incriminate any one specific variety.

Symptoms. The symptoms and signs, as may be expected, vary greatly and are often masked or obscured by a primary condition. The age and condition of the patient, the nature and severity of the predisposing factor, the variety of bacteria present and the extent of the lesion all modify the symptoms. The presence of cough, a little higher fever, pulse rate, and respiratory rate, although they may actually be caused by pneumonia, may be ignored or overlooked during some other serious illness. It may be impossible to differentiate pulmonary hypostasis or infected atelectatic areas from pneumonia. Pneumonia may be present without causing any telltale symptoms. On the other hand, pneumonia may commence suddenly and overwhelm the patient in a day or two.

In the *average case* the patient gradually or suddenly becomes worse, or does not recover from his primary disease so rapidly as would be expected, or there may even be a period of apparent recovery before new symptoms appear. Evidence of tracheitis and bronchitis with cough and sputum may first be noted or they may never appear. The pulse rate, respiratory rate, and temperature show a tendency to rise and the patient feels sick. The skin may

be hot and dry or there may be profuse sweating. Thirst may be marked. A sensation of tightness in the chest and substernal pain or oppression are often noted. The cough becomes more and more distressing and exhausting. Evidence of actual pneumonia is indicated by the severity of the constitutional reaction, an increase of the pulse rate, respiratory rate, and the temperature, and by physical and roentgenographic signs of pulmonary disease.

The *fever curve* is not uniform. It may be persistently high, but more often shows considerable variation as the process spreads from place to place in the lungs. In patients who show no frank physical signs, a diagnosis of pneumonia can often be guessed at correctly from the irregular character of the fever chart. Hyperpyrexia may occur, but in debilitated individuals the temperature may be only slightly elevated or even subnormal. The fever may last from a few days to several weeks and subsides by lysis in those who recover. Frequently, after a normal period lasting a few days, there is a recurrence of fever and symptoms. Persistence of fever indicates activity of infection. During bronchitis, any fever lasting longer than four days is suggestive of pneumonia.

The *pulse rate* is usually increased, but relatively less so than the respiration. The pulse rate curve often follows the fluctuations of the temperature curve. The rate may rise to 150 per minute. Irregularities are occasionally noted.

Respiration is affected roughly in proportion to the extent of lung involvement. The rate increases and may reach 60 to 80 per minute. Tachypnea and dyspnea may be very distressing. Cyanosis is characteristic and is usually present. In fatal cases the pulse and respiration become feeble, cyanosis and apathy increase, tracheal rales appear, and death ensues.

Cough is often troublesome. In the aged it may be ineffectual and the sputum is either not raised or is swallowed. The sputum is variable, ranging from thin mucus to tenacious mucopus. The quantity raised varies greatly. It may be pinkish, bloody, or purulent.

Pleuritis and herpes labialis occur less often than in lobar pneumonia. Frequently, vomiting and anorexia occur. The urine shows the usual changes resulting from fever.

Like the clinical course, the physical signs are variable. When considering the physical signs, it is helpful to bear in mind the underlying pathologic process. The spread of the inflammation from place to place, with increase of temperature and the development of symptoms as fresh areas become involved, is usually accompanied by an increase of abnormal physical signs, although not always so. The extent of the area over which signs are elicited may be no larger than a silver quarter or may include the greater part of the chest. Abnormal physical signs may be entirely absent or may be obscured by the primary condition (e. g., malignant metastases, infarcts, bronchiectasis, asthma). Physical signs vary from day to day, even from hour to hour, disappearing in one area to reappear later, to be followed by fresh signs elsewhere in the lungs. The spread of infection from place to place or the temporary plugging of the bronchi with exudate, atelectasis and emphysema is a factor responsible for the rapid migration and variation of physical signs.

Complications. Bronchitis is often present. Laryngitis and aphonia may be present. Endocarditis, pericarditis, emphysema, and bacteremia occur

although not so often as in pneumococcal lobar pneumonia. Multiple abscesses of the lungs and massive necrosis may follow in protracted cases. Relapse is common (See Chapter 22.)

Anemia is unusual unless it is already present or the disease runs a protracted course.

Diagnosis. In senile pneumonia the variability of causes, the insidious onset, irregular course, and uncertainty of physical signs present difficulties in diagnosis. This can often be presumed from vague suggestive signs, symptoms, or behavior of the patient and relative frequency of pneumonia in certain conditions. A roentgenogram of the chest is of great value. As mentioned previously, it is sometimes impossible to decide when bronchitis or hypostasis becomes pneumonia, but when rales, dyspnea, and cyanosis are present, when fever lasts longer than four days, and when constitutional symptoms are considerable, pneumonia must be suspected. Differentiation from typical pneumococcal lobar pneumonia is made by its insidious onset, irregularity of fever and course, the variability of the quality and location of rales, the absence of signs of frank consolidation, the infrequency of pleurisy, herpes, and rusty sputum.

The *leukocyte count* is usually increased, ranging from 15,000 to 30,000 or more. The polymorphonuclear leukocytes predominate. Leukopenia may be present in debilitated persons. The *sputum* is purulent, perhaps bloody or rusty, and contains a variety of bacteria. Cultures obtained from the lung by lung puncture during life and at the necropsy table contain mixtures of bacteria on smear or culture, so that it is impossible to decide if any one species is especially operative. Blood culture may reveal the causal agent.

The signs of atypical pneumonia may be simulated by pleural exudate, infarction, atelectasis, bronchiectasis, peripneumonitis, by the plugging of bronchi, pulmonary congestion, and by massive collapse. History, x-ray examination, removal of the plug by bronchoscopy, or an exploratory puncture will usually decide the question.

Prognosis. The data on prognosis are inaccurate because, as stated previously, it is difficult to tell whether pneumonia occurs because the patient is dying or whether the patient really dies from pneumonia. Furthermore, in many old patients, pneumonia as the cause of death may not have been recognized in the absence of roentgenographic or necropsy evidence. Nevertheless, the death rate is high. In general it is said to be about 60 per cent in patients over sixty, and 80 to 100 per cent in those over seventy. The prognosis is more favorable when pneumonia occurs secondary to an acute infection of the respiratory tract and in the absence of other serious conditions.

TREATMENT

Specific Therapy. With the possibility in mind that the causative bacteria are sensitive, penicillin or sulfadiazine should be given in adequate amounts for several days as described under Lobar Pneumonia, page 348. If gram negative bacteria predominate, streptomycin or one of the other newer antibiotic agents may be of value.

General Treatment. Since competent *nursing care* is of such great importance in the management of a pneumonia patient, it is best to employ one or more trained nurses. When financial status permits, it is wise to move the patient to a hospital. This involves practically no danger when the patient

is transported on a stretcher in a modern, warm, and comfortable ambulance. The transfer, if contemplated, should be made early in the disease, for the risk is somewhat increased in the later stages.

At the first symptom or sign of infection of the lung or of threatened pulmonary involvement, the patient should be placed in bed immediately if not already there. Efforts to provide comfort and absolute physical and mental rest are essential. Senile patients sometimes are more comfortable and do better when propped to a semisitting position or even when placed in a large upholstered chair. The patient should be turned occasionally, particularly

In general, there should

examinations are harmful. The patient should be induced to sleep as much as possible. The bedpan should be used to avoid the need for leaving the bed. The room should be lighted, warm, and well ventilated without direct cold drafts. Unless the patient is too sick, a sponge bath followed by an alcohol rub is allowed daily or every other day. For restlessness or when the fever exceeds 39°C (102°F), tepid sponge baths may be given several times daily, if agreeable to the patient. Careful attention should be given to oral hygiene. The teeth and gums should be thoroughly brushed daily and a mildly alkaline mouth wash used. The bowels may be kept open with liquid petrolatum, 15 cc to 30 cc ($\frac{1}{2}$ to 1 ounce) each night, or with an enema every other day, or with mild laxatives, if this has been the patient's habit. Drastic cathartics or purgatives are exhausting and are contraindicated.

Medication should be avoided except when absolutely necessary. Sodium citrate, 2 to 3 gm (30 to 45 grains), every two or three hours when awake, has been recommended by some to promote renal activity. Its superiority over plenty of water as a diuretic is questionable. Aromatic spirits of ammonia, 2 cc ($\frac{1}{2}$ drachm), is occasionally used for stimulation. Both drugs should be promptly discontinued if disagreeable to the stomach.

Oxygen When the smaller air passages are partially or completely plugged by edema or inflammatory exudate, a marked decrease in the oxygen tension in the alveoli occurs causing an oxygen deficit in the blood with consequent dyspnea, tachypnea, and cyanosis. For this reason oxygen has been administered with considerable success. The optimum effect from oxygen therapy is to be obtained if this form of treatment is given in the early stages of atypical pneumonia, when the edema alone is present, rather than after frank inflammatory exudate collects. Oxygen supplied under slight pressure is said to be helpful when generalized pulmonary edema is present. Those patients who are deeply cyanotic will usually show more improvement than those in whom this is not so evident. Concomitant with the improvement in the patient's color, a drop in temperature and improvement in other ways may occur. The oxygen tent with a gas saturation of between 40 to 60 per cent is the most desirable apparatus. The temperature in the tent should be regulated to the patient's comfort. Oxygen may be administered by inserting a catheter through the nares to the pharynx and allowing it to flow in at the rate of 4 to 6 liters a minute. The gas should be passed through a wash bottle of water to moisten it. Pharyngitis is apt to occur.

Diet There are usually no special rules to be observed in prescribing a diet unless diabetes or some other metabolic or nutritional disease is present. The patient should be encouraged to take as much hot, simple, and nutritious

food as can be digested. Meals should be small and frequent. Soups, broths, fruit juices, milk, and toast are usually well tolerated during the period of intoxication. Later, custards, eggs, lamb chops, and chicken may be added. Tea and coffee may be permitted, according to the patient's habits. Plenty of water should be taken. When nauseated, carbonated water or ginger ale is often agreeable. Nausea, vomiting, or flatulence necessitates the modification or temporary withholding of food, or the elimination of sugar or milk. Experimentation is necessary at times to arrange a proper diet.

The intravenous administration of 200 cc. of a 25 per cent solution of *dextrose* or of preparations of amino acids several times daily may be required if the patient cannot or will not eat. Hypodermoclysis or venoclysis of sterile isotonic solution of sodium chloride may be necessary to furnish the required amount of fluid, in doses of 500 to 1000 cc. Too much should not be given if edema threatens.

Alcoholic beverages (wine, brandy, whisky) may be given occasionally and are often beneficial as stimulants. They may be given routinely to chronic alcoholic patients and are said to prevent the development of delirium tremens.

Treatment of Special Symptoms. For *headache* an ice-bag or a hot water bottle, according to the comfort derived, may be applied to the head. A darkened, quiet room or a damp, cool cloth covering the eyes are often agreeable. Frequent cough should be relieved. Acetylsalicylic acid 0.3 to 0.6 gm (5 to 10 grains), or aminopyrine 0.18 to 0.3 gm (3 to 5 grains), may be given in one or two doses and stopped. Codeine, by mouth, 0.06 gm (1 grain) may be required.

Cough. Repeated inhalations of the vapor of tincture of benzoin compound or oil of eucalyptus from a croup kettle or the use of the plain steam tent serves to lessen cough and favor the expectoration of tenacious mucus. The administration of 5 per cent carbon dioxide by means of a mask is said to be an efficient expectorant measure. When inhalations are inadvisable or impractical, it may be necessary to administer codeine 0.06 gm (1 grain) or paregoric 2 to 4 cc ($\frac{1}{2}$ to 1 drachm) every four hours, to procure sleep and prevent exhaustion. Cough syrups containing various medicinal expectorants (ammonium chloride, ipecacuanha) were formerly widely used, but are of little value. They usually "upset the stomach" and cause nausea. The steam tent is useless when the secretions are profuse and watery. In the latter case, atropine 0.6 mg ($\frac{1}{100}$ grain), or more, every four hours has been recommended for its supposed influence in lessening secretion. Its influence on pulmonary edema and inflammatory exudates is questionable and actual harm may result. Hot wet poultices, hot camphorated oil, mustard plasters and other counterirritants and antiphlogistics are seldom used. They are troublesome, disturb the patient, and can be substituted for by the much more convenient electric pad, hot water bottle or ice-bag.

Pain in the chest is best combated with the many tailed flannel chest binder adjusted to apply pressure where needed. It is easily applied, readjusted, or removed. It should not be used unless it actually diminishes the pain. Adhesive plaster should never be used in pneumonia. An electric heating pad, hot-water bottle, or ice-bag is convenient and comfortable. For intractable pain, morphine 15 mg ($\frac{1}{4}$ grain) by hypodermic injection may be necessary. Opiates should be used as a last resort. They frequently give rise

to obstinate constipation and tympanites which are to be avoided in pneumonia Diathermy occasionally gives relief from pain Whether this form of therapy has any distinct advantage over the local application of heat by simpler means remains to be determined Roentgen ray therapy is of no avail

When anemia develops in protracted cases, treatment according to instructions given on page 204 may be used or transfusion of blood may be indicated

The *circulatory system* is best conserved by complete rest Digitalis should be given to those who have decompensated cardiac disease or to those who develop auricular fibrillation during the illness 4 to 8 cc (1 to 2 drachms) of the tincture of digitalis U S P or corresponding doses of the powdered leaf every two hours, until 12 or 16 cc (3 or 4 drachms) is given, according to the weight of the patient If necessary, the effect may then be maintained with 0.5 to 1 cc (8 to 16 minims) doses every four to six hours for a time (see page 388)

In *circulatory collapse*, indicated by lowered blood pressure, perspiration, cyanosis, and edema of the lungs, the patient should be surrounded with hot water bottles and warm blankets Oxygen should be administered Adrenalin in a concentration of $\frac{1}{1000}$ may be given in 0.5 cc (8 minims) doses every twenty minutes, for 5 or 6 doses The intravenous injection of 500 to 1000 cc of 5 or 10 per cent solution of dextrose or sucrose may be helpful A hot mustard foot bath or the removal of 200 to 500 cc of blood by venesection is occasionally of value in plethoric individuals Stimulants, such as alcohol, hot strong coffee, or aromatic spirits of ammonia, may be used Caffeine may be helpful, but its effects in preventing sleep when sleep is greatly needed must be considered Camphor and strychnine are ineffective According to recent opinion the hypodermic injection of morphine sulfate 0.01 to 0.015 gm ($\frac{1}{8}$ to $\frac{1}{4}$ grain) is most effective

Abdominal Distention The less obstinate forms of colonic distention can often be relieved by a mild laxative or enema The insertion of a rectal tube is often helpful Hot stupes to which turpentine has been added (1000 cc of water and 10 to 20 cc turpentine) for further counterirritant effect may be applied over the abdomen It is questionable whether this rather trouble some method has any advantage over the simpler use of an electric heating pad or hot-water bottle Drugs like pituitrin (solution of posterior pituitary, U S P, 1 cc given subcutaneously or intramuscularly) are said to be helpful The use of the continuous suction apparatus as designed by Wangenstein has been used with success for upper intestinal or stomach distention or the Miller-Abbott tube may be used, or a tube may be inserted into the sigmoid or colon for colonic distention

Hiccough The simplest methods employed to control hiccough are counterirritants in the form of ointments containing volatile oils such as peppermint, methyl salicylate, menthol, or capsicum applied to the skin of the chest or neck, or carminatives such as peppermint water, 4 cc (1 drachm) chloroform, 0.06 cc (1 minim), whisky, brandy or rum, 4 to 8 cc (1 to 2 drachms), given by mouth Plain iced or carbonated water is sometimes helpful Ethyl chloride has been sprayed on the skin over the areas where the diaphragm is inserted The stomach may be washed out, or its contents aspirated, if it is distended with gas Carbon dioxide gas in 5 per cent concentration mixed with air or oxygen and given with a mask inhaler may

increase the depth of respiration and relieve hiccough. Oxygen inhalation to relieve anoxemia may be helpful.

Sedatives such as sodium bromide, 1 gm (15 grains), chloral hydrate 1 gm (15 grains), or phenobarbital, 0.06 to 0.2 gm (1 to 3 grains), should be tried. Morphine, 15 mg ($\frac{1}{4}$ grain), may sometimes be necessary to permit a few hours rest. As a last resort, injection of alcohol into the phrenic nerve may be tried.

Diversion or concentration of the patient's interest on other matters, as by pulling or depressing the tongue, may be helpful in patients not too ill. Hiccough often stops spontaneously.

Restlessness and Delirium. Thoracic pain, cough, "toxemia," and apprehension are the chief causes of restlessness and insomnia in pneumonia. The treatment of some of these factors has already been outlined. Frequently the milder sedatives such as sodium or potassium bromide, 1 gm (15 grains), or phenobarbital, 0.09 gm ($1\frac{1}{4}$ grains), are sufficient to quiet the patient. Occasionally the oxygen tent is helpful. In the occasional case, however, morphine alone, in doses of 8 to 15 mg ($\frac{1}{8}$ to $\frac{1}{4}$ grain), hypodermically, is effective. A single dose may suffice, but judgment is required if more is needed. Headache and other aching pains may be relieved by acetylsalicylic acid, 0.3 to 1 gm (5 to 15 grains), or aminopyrine, 0.3 gm (5 grains), repeated as required.

The development of delirium requires constant nursing attendance lest the patient harm himself or others. Morphine under these circumstances may aggravate the excitement and must be used with caution. Persuasion should be tried first. Simple restraint by fastening down the sheets, or with anklets or cuffs, may be required. Sodium amytal sterile solution in ampules may be administered intravenously in similar dosage to delirious patients who resist oral medication or to those in whom a prompt effect is imperative. The barbiturates, however, may cause severe depression in senile patients. Hydrotherapy, in the form of tepid or cool sponge baths, is helpful.

Patients known to be addicted to the use of alcohol should be given small amounts of whisky or brandy, 4 to 15 cc (1 to 4 drachms) at four to six hour intervals, as a possible preventive of delirium tremens.

Complications. The most important and commonest complication is empyema which occurs more frequently in old age than in youth (see page 339). Other focal pneumonia may also occur.

Prognosis. The prognosis is usually good, but it is not infrequently fatal in old age.

PNEUMOCOCCAL LOBAR PNEUMONIA

The typical course of pneumococcal lobar pneumonia with sudden onset, a shaking chill, pain in the chest, cough, rusty sputum, and high fever is adequately discussed in all textbooks of medicine and needs no detailed repetition here, since its behavior in old age is occasionally typical. Certain characteristics of the disease peculiar to senility do, however, exist and warrant discussion.

Etiology. The pneumococcus is the cause of clinically typical lobar pneumonia in the majority of cases, but the incidence of distribution of the various types is somewhat different than in youth, as shown in the following table

Age	Under 50	Over 60
Type I	36 per cent	20 per cent
Type II	12 per cent	14 per cent
Type III	11 per cent	34 per cent
Other Types	33 per cent	34 per cent

Type I, which usually is associated with typical lobar pneumonia, is found less often and Type III pneumonia considerably more often after the age of sixty. In one series about 50 per cent of cases of Type III pneumonia occurred in persons more than fifty-five, and nearly twice as often in men as in women. Seventy per cent of patients older than sixty years had Type III pneumonia.

Pneumonia caused by pneumococci of Types I and II is usually an exogenous infection contracted from without. Pneumonia due to pneumococci of all other types, of which there are more than thirty-two, usually is an autogenous infection; that is, these pneumococci are often present in persons as saprophytes. They seldom give rise to typical clinical lobar pneumonia in the aged. In any case some actual predisposing cause is evident which serves to permit invasion of the pneumococci and the ensuing disease. Predisposing causes are detectable in about 75 per cent of cases and are chiefly mild infections of the respiratory tract, such as colds, grippe, or influenza (50 per cent) and exposure, exhaustion, and acute alcoholism (17 per cent). Chronic disease of the pulmonary or vascular systems and alcoholism are often present.

Symptoms. The typical onset of pneumococcal lobar pneumonia, its subsequent course and abrupt termination, is less often seen in old age than in youth. Typically, after some mild indisposition, there is a sudden onset with a shaking chill, stabbing pain in the chest, cough, rusty sputum, and high fever. The number of leukocytes is usually elevated to 15,000 or 20,000. Cyanosis, dyspnea, and tachycardia occur. In favorable cases, after a period of several days or a week, recovery commences and the temperature declines slowly. In the aged, crisis occurs in only about 10 per cent of cases.

The *physical signs* in the early stage are those of congestion with impaired resonance, diminished tactile fremitus, rales, and suppressed breath and voice sounds. When the lung is consolidated there are increased tactile fremitus, dullness, bronchial breath sounds, egophony, and pectoriloquy. During resolution, these findings disappear and rales are again evident.

Prognosis. The high mortality of pneumonia in the aged is common knowledge. It is substantiated in the following table (Heffron) ³

Age Group	Percentage Mortality
2-10	6
20-29	16
40-49	42
50-59	66
60-69	66
70-	82

The severity of the predisposing disease or condition, the presence of chronic disease, the type of the pneumococcus (II, III or V especially), bacteremia, the presence of complications, and the poor general condition of the patient are all unfavorable factors in prognosis. The mortality rate is higher when pneumonia assumes an atypical form than in frank lobar pneumonia.

Diagnosis. It is desirable to determine the etiologic agent in every case of pneumonia. Sputum, or material obtained by swabbing the larynx, should be examined by a stained preparation and plated on blood agar. The Neufeld technic should be used to identify the type of pneumococcus present. Mouse inoculation may have to be used when pneumococci cannot be found otherwise. Blood cultures should be made at the earliest moment possible.

Roentgenograms of the chest are particularly useful in diagnosis. If a portable apparatus is used, a plate can often be made with less fatigue to the patient than a careful physical examination.

Treatment. The general treatment is the same as that given previously (pp 344-348).

Specific Therapy. As soon as the diagnosis is made and material has been collected for study, penicillin should be injected intramuscularly at intervals of twelve hours in doses of 100,000 units. The initial dose should be doubled. More may be needed on occasions or the first dose may be injected intravenously. Treatment should be continued until the temperature has been normal for several days. Relapse may require resumption of treatment. Oral medication is not reliable enough to be depended upon. If it is used, the dosage should be much greater, that is, at least 150,000 units every three hours.

If penicillin is unavailable or if the patient is sensitive to it, sulfadiazine may be given orally, 4 gm initially and 1 gm every four hours thereafter, together with enough sodium bicarbonate to keep the urine alkaline. If oral therapy cannot be used, sodium sulfadiazine may be given intravenously or subcutaneously in the same amounts. Since sulfonamide compounds may be excreted poorly in old age, the amount in the blood must be measured occasionally to avoid overdosage. Levels between 4 and 10 mg per 100 cc of blood are desired. The leukocytes must be counted frequently lest leukopenia occur. The urine must be examined to detect red blood cells and the patient constantly observed to detect jaundice, erythema, or purpura, which necessitates stopping the drug.

The decision to use or not to use penicillin or sulfadiazine is particularly difficult in geriatric practice. Obviously there is no need to use it if the infection is not caused by bacteria susceptible to its effects, but in old persons it

one is reasonable to start it if some other disease not amenable to the drugs. In the case of clear-cut clinical lobar pneumonia, the decision is simple and the drug should be employed in the usual dosage, due attention being given to proper elimination.

Serotherapy. There is scarcely any use for serotherapy at present. Under unusual circumstances, however, it may at times still find a place. If so, the causative pneumococcus must be accurately typed and specific concentrated rabbit antipneumococcus serum used, testing the patient for sensitivity to serum in the standard manner. An initial dose of 50,000 to 100,000 units

intravenously is recommended and thereafter in 20,000 unit doses every six hours according to the need, until recovery occurs

Pleurisy. Pleurisy occurs with such regularity in lobar pneumonia that it should be considered as part of the disease rather than as a complication. Pleurisy may be a primary condition in the absence of pneumonia. Often an effusion of sterile fluid may be large enough to cause embarrassment of breathing or of the heart and will require removal by aspiration. In most cases it disappears spontaneously.

Empyema. In about 8 per cent of all untreated pneumonia, especially in those with early severe and constant pleuritis, the fluid is infected with pneumococci, and occasionally with other cocci. Thus the pleuritis becomes empyema. Empyema may occur during or after the pneumonia, or as an apparent primary infection in the absence of pneumonia. Pneumococci of types I and V seem especially apt to cause empyema. The fluid is usually thin at first but gradually becomes thicker until greenish yellow pus forms.

Diagnosis. Empyema should be suspected when improvement fails to occur at the usual time, when severe pain in the chest and sweating are present, when physical signs of fluid appear or when the temperature becomes remittent. On the other hand there may be no clinical symptoms or signs of empyema at all and it may be discovered by roentgenography or at necropsy. If there is any doubt as to the presence of pus, exploratory aspiration should be performed, for diagnosis and culture. The mortality rate in untreated cases is about 40 per cent.

Treatment. Modern treatment of pneumonia has greatly reduced the incidence of empyema. When it does occur, pus should be aspirated and 100,000 units of penicillin should be injected into the empyema cavity about once a day. The duration of treatment depends upon the clinical response. Treatment should be continued for a week or more after apparent recovery lest relapse occur. If no benefit is evident, surgical thoracotomy and drainage is needed. The optimal time to operate is a matter of judgment—not too soon and not too late. Undue delay in operating prolongs the illness and may permit further spread of the infection, emaciation, anemia, and other complications. The operation itself should provide free and open drainage. This can be accomplished only by thoracotomy with rib resection and the insertion of large-bore drainage tubes. In competent surgical hands this procedure can be done under local anesthesia. Drainage must be adequate to prevent the development of walled off pockets.

It seems scarcely necessary to mention the great importance of maintaining the patient's nutrition with a *high caloric diet* (3000 to 4000 calories) adequate in vitamins (see page 189). Blood transfusions are often necessary, when illness is prolonged. Fresh air, a cheerful environment, and sunshine are conducive to rapid convalescence.

PREVENTION OF PNEUMONIA

General Measures. The greatest factors in the prevention of pneumonia in the aged are the maintenance of good health and all that this implies, preventing or minimizing other illness or conditions which predispose to pneumonia, and the avoidance of mild infection of the respiratory tract, such as colds. These factors are for the most part discussed elsewhere. As much

activity as is consistent with the person's general condition is advised. Old persons should not be confined to bed unless it is absolutely necessary. The movements and exercise incident to sitting up in a comfortable chair favor respiratory motions which in turn assist in maintaining the pulmonary system in a more normal condition. Inactivity results in lack of aeration of the deeper portions of the lungs, lessened diaphragmatic excursion, and the development of hypostasis and patches of atelectasis. These conditions favor the invasion of bacteria and the development of pneumonia. If the patients are necessarily confined to bed, their position should be changed frequently to prevent hypostasis, and deep breathing exercises should be ordered at regulated intervals of several hours during the day.

The patients' surroundings should be cheery, warm, light, and well ventilated but free from drafts. If the financial status allows, migration to a warm climate in winter is beneficial.

Since pneumonia is so often ushered in by a minor infection of the respiratory tract, it is of utmost importance to protect the aged from contact infection. No person with colds, sinusitis, or sore throat should be permitted to come into the room. If this is unavoidable, face masks of several layers of fine gauze covering the nose and mouth may be worn both by the aged person and the one who carries the infection. The value of this practice, however, is not established. Viruses and bacteria when dry float about in the air much like smoke and may pass through the finest fabric filters.

If the aged one does develop an infection of the upper respiratory tract, treatment such as described on page 309 should be begun immediately. The question has recently arisen as to whether persons with such infection should be given penicillin or sulfadiazine in order to prevent pneumonia. I have opposed this practice in general and do not believe it should be applied in geriatric medicine, unless evidence gathered in controlled studies eventually proves it to be worth while, or unless an epidemic of pneumococcal or hemolytic streptococcal pneumonia exists in the environment. If the patient is under careful observation, penicillin or sulfonamide chemotherapy should be reserved until evidence of pneumonia caused by bacteria amenable to its action appears, at which time it should be applied thoroughly. Many forms of pneumonia occur which are not influenced by the therapeutic agents available at present.

Specific Prophylactic Vaccination The value of various forms of vaccine prophylaxis against pneumococcal pneumonia is not established. Experiments are under way at present with the use of pneumococcal capsular polysaccharide as an antigen. According to Kaufman¹ although the injections of this substance seemed to reduce the incidence of pneumonia in the group of old persons he studied, there was no appreciable effect on the course of the pneumonia when it did occur or on the case fatality rate.

OTHER SPECIFIC FORMS OF PNEUMONIA

Hemolytic Streptococcal Pneumonia This form of pneumonia may be primary or secondary to some other mild respiratory tract infection, especially sore throat. The onset may be sudden but usually it merges with the preceding infection. The temperature, pulse rate, and respiratory rate increase, cough, often with bloody sputum, appears, the leukocytes increase and signs of atypical or 'bronchopneumonia' appear (p. 341). *Diagnosis is*

made by the predominance of hemolytic streptococci in the sputum or by their presence in the blood or in focal purulent collections. The mortality rate in the aged is high, perhaps more than 80 per cent. The most frequent

be anticipated

Penicillin or sulfadiazine should be given in the manner as prescribed on p 350. Although studies⁴ show that the mortality rate seems to be reduced by chemotherapy, the results are not particularly satisfactory, especially in old patients.

Hemolytic Streptococcus Empyema. This frequent complication of hemolytic streptococcal pneumonia is often heralded by severe pleuritic pain and friction quickly followed by massive effusion which tends to reaccumulate as rapidly as it is drained. The fluid is serosanguineous at first and may contain many streptococci, later it tends to become purulent. Effusion is often first discovered by roentgenography.

Treatment Sulfonamide is given, but here again the results are not always satisfactory.⁴ Specific treatment is the same as advised for pneumococcal empyema. Aspiration of the fluid at intervals, especially if respiration or the circulation is embarrassed, is advised. For treatment of pain see p 346. In many cases it becomes necessary to perform a thoracotomy, but it is important to delay operation until the most severe stage of the infection has passed. Because of the prolonged course of the disease, particular attention must be given to maintaining an adequate diet. Transfusion of blood may be necessary for the anemia incident to the disease, to chemotherapy, or to both.

staphylococcal infections elsewhere in the body, the predominance of staphylococci in the sputum, their presence in the blood or in focal purulent lesions, and the development of multiple abscesses of the lung, if the patient survives long enough. The mortality rate in the aged is high.

Treatment Penicillin in doses of 200,000 units daily or more as recommended on page 350 is indicated.

Bacillus Mucosus Capsulatus (Friedlander Bacillus) Pneumonia.⁵ This form of pneumonia is uncommon but occurs chiefly after late middle life. For some unknown reason it is far more common in men than in women. Predisposing factors are numerous: chronic alcoholism, debility, and chronic pulmonary disease are outstanding in importance.

The onset of the disease, as in other pneumonias of old age, may be sudden like that of typical lobar pneumonia but is more often gradual. Bright red sputum or actual hemoptysis is a characteristic feature. Prostration, nausea, vomiting, diarrhea, delirium, dyspnea, and cyanosis are frequent. Fever may be high, or the whole course of the disease may be asthenic in form.

Chronic Form In rare cases, if death does not occur, the disease may become chronic, lasting for several weeks or months. The clinical course then resembles that of pulmonary tuberculosis, and has been mistaken for it.

especially when the upper lobes are involved Abscess formation, necrosis, and cavitation occur Empyema, pericarditis, and meningitis may follow

Diagnosis Diagnosis is made by the demonstration of the causative bacilli in the sputum, pus or blood The bacilli may be identified on culture and by inoculation of mice They are also classified as to type, type A causes the majority of cases The physical signs may be those of consolidation of lobes, but in the aged they are more often those of atypical pneumonia If the course lasts long enough, necrosis with formation of cavities and fibrosis occurs Roentgenograms show dense infiltration and perhaps cavitation Bacteremia occurs in the majority of cases The leukocyte count is variable The sputum typically is bloody, brick red, or brown, and slimy, later becoming purulent

The mortality rate is over 80 per cent in the aged

Treatment The futility of available forms of treatment is indicated by the high mortality rate Treatment is still largely symptomatic At present, streptomycin given intramuscularly in doses of 1 gm every six hours seems to be helpful It has also given good results in pneumonia caused by *Hemophilus influenzae* Specific immune serum, penicillin, and sulfonamide compounds have not been of value

Influenza and Influenza-Like Diseases. "Viral" Pneumonia. According to recent studies, influenza virus and other similar filtrable viruses, although they cause mild infection of the respiratory tract in the majority of cases, may actually cause specific forms of pneumonia The pneumonia may be mild or severe without the agency of bacteria In many cases, however, the viral infection seems to permit the invasion of bacteria, which in turn cause pneumonia with characteristics modified by the bacterium most active⁶

In uncomplicated 'viral' pneumonia, the symptoms are quite typical The onset is often gradual, merging with preexistent nasopharyngitis The temperature rises and symptoms become intensified There are chilly sensations, unproductive hacking cough, sweating, headache, photophobia, and relative bradycardia, and in some cases dyspnea and cyanosis Evidence of pneumonia, usually diffuse and bilateral, is often detected first by roentgenography, since abnormal physical signs may be delayed or are minimal There is seldom any sputum and the leukocyte count is normal (see Fig 68)

In enfeebled persons, the symptoms are so atypical and masked by the asthenic course that pneumonia may be unsuspected during life, or discovered only in roentgenograms

In robust old folk, the mortality rate is low, if no other disease or complications are present

The treatment is supportive and symptomatic as described on page 346 Chemotherapy and antibiotic agents are of no value in treatment in uncomplicated cases

Diagnosis During an epidemic the diagnosis is comparatively easy and is made on the basis of the clinical behavior, the normal leukocyte count, and roentgenographic evidence of pulmonary invasion Diagnosis in sporadic cases may be difficult because of the insidious nature which may be characteristic of any form of pneumonia in old persons The matter is of especial importance with reference to chemotherapy Neither penicillin nor the sulfonamide drugs have a beneficial effect on "viral" pneumonia and should not be used unless certain bacteria, such as pneumococci or hemolytic strep-

cocci, become secondarily invasive. Under some circumstances it may be desirable to use penicillin or sulfadiazine anyway, but if no beneficial effect is noted in twenty four or forty eight hours it should be discontinued.

Acute Tuberculous Pneumonia Although it is a rather rare disease, acute tuberculous pneumonia may occur at any age. It usually develops in persons

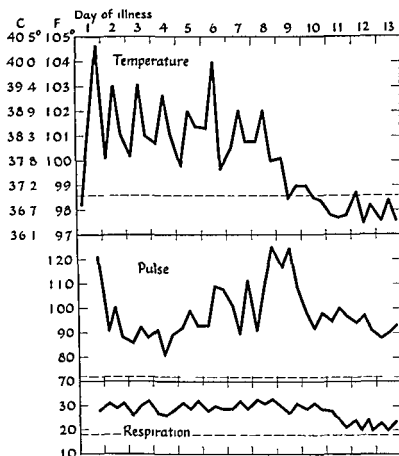


Fig 68 Viral pneumonia. A woman aged seventy six noted dryness of the throat and cough March 16. The next day the temperature rose abruptly to 40.3°C (104.5°F) and she became very sick, confused, somnolent and had unproductive cough. Physical signs were those of diffuse bronchitis and pneumonitis. There was relative bradycardia in the early period. Cyanosis, dyspnea, and profuse perspiration were present. The temperature gradually declined and recovery took place on the tenth day. Four other members of the patient's family were sick with a similar although milder infection at nearly the same time.

who have had chronic pulmonary tuberculosis and is probably the result of a sudden increase in the number of bacilli in the lung. The focus but diffuse and signs of consolidation develop which later change as softening and cavity

formation occur. In the *diffuse form* the onset is gradual, with signs manifest in various portions of the lungs. In both forms the course is usually rapidly downward, with remittent fever, sweats, toxemia, and emaciation. The sputum is purulent, often blood tinged. The mortality rate is high. In rare cases the disease becomes chronic (see p. 324).

Diagnosis The diagnosis in old persons is often confused with other forms of pneumonia, until tubercle bacilli are found in the sputum. Bacterial pneumonia may coexist with pulmonary tuberculosis. A history of previous attacks of tuberculosis, or a long period of failing health or of contact with the disease, is of importance. Roentgenograms are of value in diagnosis.

Treatment The treatment is symptomatic as described on page 330. Recent study shows that streptomycin in large dosage over a long period is indicated but information is still incomplete.

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SECTION V

DISORDERS OF THE CIRCULATORY SYSTEM

CHAPTER 24

THE NORMAL SENILE HEART

HOWARD B. SPRAGUE

In the study of that complex organism—Man—there is no more difficult decision than the limits of normal throughout a lifetime. A consideration of this problem in the cardiovascular field is of primary importance since it is in the *circulatory system* that we look for the *major senescent changes* determining longevity.

In the business world the prudent manager sets up reserves for depreciations which are at different rates for different fixed assets. So in man the tissues of different individuals age at different rates and, at best, we can but estimate at what age a man's heart and blood vessels will have exceeded their *useful life*. The term "useful life" is chosen with intent since mere attainment of great years may bring only sorrow and chronic invalidism, and the heart may continue to beat when it is nourishing a framework from which even the will to live has disappeared. In this selective survival hereditary influences play an important part.

The heart is the final arbiter of life and its integrity depends on its coronary supply as related to the demands put upon it. Normal age changes start at least at birth and must be accepted. Premature or accelerated aging by the mechanism of vascular occlusion introduces the lethal pattern. The fundamental cause of such atherosclerotic or arteriosclerotic vascular occlusion is unknown. In describing its effects upon the myocardium, however, it is not satisfactory to use the term "myocarditis," which implies an inflammatory condition of heart muscle. Myocardial ischemia, with varying degrees of myocardial infarction, and replacement fibrosis are the actual changes.

Since aging is a continuum starting at conception and developing with periods of greater or lesser acceleration throughout life, there must be an infinite series of normals against which to compare any one of us from year to year. There are no accurate techniques for such vital titration which can be applied to man, however. If one observes an individual in the senescent period after a lapse of a few years, one may say that he has aged, but so indeed has the observer, and another subjective reference point is introduced in addition to the objective normal for that age period. Particularly is it true that the assessment of normality in the cardiovascular system remains difficult and often so. The real

age, body type, and condition of muscular fitness?" A practical corollary to this, which is not based on physical or instrumental examination, is 'Has this person angina pectoris?'

PATTERN OF NORMAL CARDIOVASCULAR AGING

General Structural and Functional Changes The normal anatomic processes of aging have been considered in previous chapters. In the heart they are, in the main, dehydration, loss of ability for cellular replacement, and vascular degeneration (See Chapter 4)

Cohn¹ has summarized these as follows: (1) increase in the subpericardial fat, (2) more rigidity and less accurate closing of the valves, (3) thickening of the endocardium, especially in the left auricle, but also on the septal side of the left ventricle and apices of the papillary muscles, (4) changes in striated muscle fibers—in advanced age the striations disappear about the nuclei (it is possible also that there are changes in the intercalated disks), (5) steadily increasing pigment at the poles of the muscular nuclei with age, (6) increase in size of the nuclei, which become more complex and take on more dye than in youth, (7) changes in the elastic tissue of the aorta, ventricles, and particularly the auricles, (8) changes in the coronary arteries, the anterior descending branch maturing the earliest, and changes in the posterior descending branch lagging, perhaps for from five to ten years, (9) decrease in the rate of the heart, (10) possibly, changes in rhythm, such as premature beats and paroxysmal auricular fibrillation, (11) decrease in ability of the heart to consume oxygen, (12) changes in the innervation of the heart, which becomes less sensitive to the effects of atropine and more sensitive to carotid sinus stimulation, (13) electrocardiographic changes (discussed later), and finally, (14) peripheral arterial and capillary changes.

Symptoms of the Aging Heart Except for Adams-Stokes attacks, and the discomfort produced by abnormal cardiac rhythms, such as premature beats, paroxysmal tachycardia, and paroxysmal auricular fibrillation or flutter, the heart symptoms bringing the aging person to the doctor are the manifestations of heart failure. To say that we must wait for the appearance of these signals is to reject all of the philosophy of preventive geriatrics. This concept of the prevention of the premature results of aging, or at least of guidance of the aging individual, is the real challenge to gerontology. Not only must there be an attempt to discover the causes of accelerated senescence, whether considered a disease or not, but also an effort to discover in advance whether or not there be an abnormal reduction in functional ability prior to organ failure. Structural change of itself does not necessarily parallel functional disturbance (See Chapter 6)

So far as the heart is concerned, such a decision can more often be arrived at by questioning the patient than by any other method or test. Yet even this statement presents a paradox since our interrogation of the patient can really only discover the *symptoms* of inducible heart failure, that is, breathlessness or anginal pain. We may, it is true, evoke a story of easily

of inactivity to a brief attempt at strenuous exertion brought on unusual dyspnea, tachycardia, or substernal pressure. Such a history warrants further

observation and perhaps electrocardiographic study, but often the factors of an increased body weight, excessive use of tobacco, and general lack of physical training account for this effort syndrome

Dyspnea produced by exertion considered inadequate by the patient, or breathlessness prolonged beyond the ordinary recovery period, should be suspected as of cardiac origin, as should any type of chest or arm discomfort having a repeated relationship to exertion or emotion

In those in the older age groups, such breathlessness may be the result of chronic emphysema and is often associated with cyanosis of the lips and fingers without cardiac disease. The absence of orthopnea in this condition and especially the finding of a heart of normal size are the most important differential points (See Chapter 22)

otherwise. Sometimes unimportant arrhythmias are the cause but, if possible, the attack itself should be observed by the physician before a definite decision is made

Possible gallbladder disease should be investigated and also careful x ray examination should be made to rule out diaphragmatic hernia. It is well to remember, however, that these conditions may be complicated by angina pectoris or coronary disease

In a series of 242 cases,² the correlation between the degree of coronary sclerosis shown at autopsy and symptoms during life has shown that with marked, moderate, and mild coronary sclerosis only 14.9 per cent had cardiac pain. The symptoms observed were as follows: palpitation, 11, precordial discomfort, 6, precordial pain, 37, dyspnea, 73, and evidence of congestive failure, 162. In those analyzed for the presence of cardiac pain during life, 91.7 per cent showed severe coronary sclerosis, and two patients had moderate sclerosis. There was no patient with mild sclerosis who had cardiac pain. In coronary heart disease, therefore, congestive failure is still much more common than angina pectoris.

Signs of the Aging Heart. *Physical Examination for Heart Size.* There is some difference of opinion concerning the normal alteration in weight of the heart occurring with age. Autopsy studies may be difficult to correlate with clinical histories, and the complete story of blood pressure changes in the subjects may be unobtainable. A recent investigation of male human hearts³ presents the formula for expected normal weight of the heart in grams

extreme old age. At least, hypertrophy is apparently a manifestation of disease. Roesler⁴ states that atrophy of the heart normally begins between the ages of sixty and sixty-five years, and may result in a decrease in weight of as much as 50 gm.

Such a statement is consistent with the observation that those individuals, without hypertension, who exceed the average life expectancy, and particularly the very aged, usually have small hearts, slow heart rates and no, or insignificant, cardiac murmurs.

The "Nomenclature and Criteria for Diagnosis of Diseases of

Heart"⁵ states that "the predictable normal weight of a heart in grams may be calculated by multiplying the weight of the body in kilograms by 4.3 or 4 for males and females respectively." In the section on x-rays of this publication there is further discussion of heart size. So far as clinical examination is concerned, it is best determined by locating the maximal apex beat. If this lies within the midclavicular line and the left border of dullness, by percussion, corresponds, the heart is considered to be within normal limits of size. Efforts should, of course, be made to rule out abnormal dullness to the right of the sternum, to the left of the sternum in the second and third interspaces, and in the region of the great vessels.

Clinical Examination of the Heart The clinical examination of the heart may tell little about its aging. Demonstrable hypertrophy may reasonably be considered evidence of cardiac strain. As age progresses, the commonest cause of cardiac enlargement is hypertension. On the other hand, high degrees of coronary sclerosis, and even myocardial infarction, may be found in normal sized hearts. The *rhythm* of the heart should be normal except for occasional extrasystoles. In childhood, sinus arrhythmia is to be expected, but it decreases with age, until in adult life it is not obvious except on forced deep breathing. The reappearance of a high degree of sinus arrhythmia in the aged person is abnormal, and suggests a circulatory disorder of the sinoauricular node. The carotid sinus reflex normally becomes more sensitive with age, but a degree of over-reactivity to the point of easily induced syncope is an abnormal mechanism which has been considered indicative of coronary disease. There is a tendency for *premature beats* (extrasystoles) to increase in frequency with age. This is of no moment. If, however, they become very numerous, originate from multiple foci in the ventricle, are produced, or increased in frequency, by exercise, or occur in a fixed grouping (not due to digitalis) they may indicate an irritable myocardial focus, caused by local ischemia, on a vascular basis. Auricular premature beats may be slightly more significant, as precursors of auricular fibrillation, than are ventricular premature beats. Care should be taken to rule out premature beats produced by digitalis and by psychic causes.

The significance of the isolated appearance of *multiple premature beats*, or auricular paroxysmal tachycardia in middle or old age, in persons not susceptible in earlier life, is difficult to evaluate. In general, it may be said that failure to abolish the arrhythmia by a vacation with increased outdoor exercise, omission of coffee, tea, Coca-cola, and other caffeine containing beverages and tobacco, alleviation of nervous fatigue, and removal of foci of infection, is in favor of a mild degenerative background for the irregularity. The same statement applies to paroxysmal auricular fibrillation or flutter, except that it should be viewed with more suspicion. Thyrotoxicosis and unexpected rheumatic mitral disease should always be ruled out as causes for paroxysmal auricular fibrillation. Paroxysmal ventricular tachycardia is almost invariably evidence of serious heart disease or of digitalis intoxication. Exceptions to this occur in adolescence and early adult life.

The *heart sounds* give little information in the absence of cardiac enlargement or hypertension. They are often normally decreased in loudness in extreme old age, but then, as at any age, the development of emphysema may be a factor in suppressing the conduction of cardiac sounds. The marked decrease in, or disappearance of, the aortic second sound associated with a

systolic murmur at the aortic area is diagnostic of calcific aortic stenosis in a person, especially a male, past middle life. This condition is frequently missed in the aged, although it is usually accompanied by definite left ventricular enlargement. Increase in loudness of the aortic second sound, or change to tambour quality, suggests hypertension, syphilitic aortitis, or atherosclerosis of the aorta. An increased pulmonic second sound suggests pulmonary hypertension.

Heart Murmurs in the Normal Aging Heart Systolic murmurs of certain types are normal from childhood through early adult life. The commonest of these is the *pulmonic systolic murmur*, varying greatly with different phases of respiration, being maximal in full expiration and minimal or absent in full inspiration. This is produced by the eddies in the first portion of the pulmonary artery which, lying anterior and in apposition with the chest wall, transmits the vibrations to the surface of the thorax. With age, the thoracic cage deepens and becomes more rigid. The heart tends to become more horizontal and this murmur disappears, although it may be brought out by exercise and is best heard with the subject's breath held at full expiration. More usual, in later life, is a *systolic murmur at the aortic area*. Cossio has explained this change in murmurs by showing that the more vertical position of the heart in youth produces a kinking of the pulmonary arc, while with the more horizontal position of the heart in later life there is a relatively greater kinking of the aorta. The normal lengthening and tortuosity of the aorta which occurs with age may also play a part in this physiologic murmur. An increase in loudness of the aortic systolic murmur should raise a suspicion of aortic stenosis or arterial hypertension—in the former case the aortic second sound is diminished or absent, in the latter, it is increased and ringing.

Cardiorespiratory systolic murmurs are found at all ages but more commonly in the young. They are apical or indefinitely precordial and are produced by displacement of air in the lung. They vary with respiration and with the position of the patient, often disappearing on full inspiration. A useful diagnostic point in evaluating such a murmur is that if the subject breathes deeply and slowly during auscultation, a point will often be found in the respiratory cycle where the murmur suddenly disappears for a few beats.

Apical systolic murmurs are sometimes as hard to assess in middle and old age as they are in childhood. The development of a definite persistent apical systolic murmur cannot be considered a phenomenon of normal aging. Usually, it signifies left ventricular dilatation, and rarely calcareous changes in the mitral valve. It may appear first after infarction of the papillary muscle of the mitral valve.

No diastolic murmur is normal. It must be borne in mind that a mitral diastolic murmur, signifying rheumatic mitral stenosis, may be found in persons over the age of eighty. Aortic diastolic murmurs occasionally develop in hypertensive patients on the basis of a dilated aortic ring and/or, in rare instances, a senile ectasis of the aorta due to an abnormal degree of loss of aortic elasticity can produce such a disturbance of valve mechanics that a loud aortic diastolic murmur may appear in the absence of lues or aortic atherosclerosis.

Blood Pressure The development of hypertension cannot be considered

a normal concomitant of the aging process. On the other hand, the existence of hypertension need not lead to premature degeneration of the coronary vascular system so far as function is concerned, nor to a shortening of life expectancy. Every physician has patients living well into the eighties, and sometimes beyond, who seem to exist for years in a friendly symbiotic relationship to their high blood pressures. In general, however, hypertension is objective evidence of disease, and as such, reference to it may be omitted here in a discussion of the normal cardiovascular system. (See Chapter 30.)

However, it seems evident that the development of some degree of systolic hypertension after the age of sixty five is so common as to have little relationship to longevity. Russek et al.^{6, 7, 8} showed in a group of retired seamen (aged 60 to 95) that average systolic and pulse pressures increase with age, while average diastolic pressure shows little variation after the age of sixty five years. Normal diastolic pressure tends to fall with age, and the number of subjects with diastolic pressures under 70 mm of mercury increases. Life expectancy is very significantly shortened by diastolic hypertension, but "for practical purposes, the clinician may regard systolic hypertension in the aged as normal." Of 3691 white males, aged fifty to ninety five years, about one third of the subjects over sixty had systolic hypertension.

Recent direct blood pressure recording studies by Bordley⁹ indicate the possibilities for error in blood pressure determinations in people with heavy or thin arms. In the former, the clinical method may, in rare instances, record the systolic pressure as much as 40 mm too high, and in the latter, as much as 40 mm too low.

X-ray Examination The roentgen examination of the heart gives us information concerning (1) heart size, (2) heart shape, (3) heart pulsations, (4) calcification of valves and valve rings (annulus fibrosus), (5) calcification of coronary arteries, (6) calcification of pericardium, (7) size, shape, pulsation, and calcification of aorta.

HEART SIZE As noted previously, the heart normally begins to atrophy in the seventh decade. Serial films showing increase in heart size as the years go on give clear evidence of cardiac pathology. In most instances, hypertension is the cause. Rarely there is a slowly developing aortic stenosis. Coronary artery disease producing multiple areas of myocardial ischemia and fibrosis with cardiac enlargement can occur without the anginal syndrome, but an electrocardiogram should help to clarify the diagnosis in such cases. High degrees of coronary artery occlusion can, however, be found at autopsy in the absence of cardiac enlargement, or even of cardiac symptoms. There is no completely reliable method of correlating heart size by x rays with other body variables to arrive at a decision about normal heart measurements in an individual case. One of the best methods, however, is that proposed by Ungerleider and Clark.¹⁰ By this method, the predicted total transverse cardiac diameter in the seven foot x ray film is correlated with the height and weight of the subject. This correlation is preferable to the older cardiothoracic ratio which relates the total transverse diameter of the heart to the internal diameter of the thorax. Whenever possible, however, teleroentgenography should be supplemented by fluoroscopy.

HEART SHAPE With advancing age the youthful preponderance of the right ventricle is overcome by the heavier left ventricle and one should expect some prominence of the left ventricle as a normal change. The heart is more

horizontal, also, largely due to a higher diaphragm so common with the increase in body weight after the thirties. In the thin aged individual, on the other hand, and especially if emphysema is present, the diaphragm will be low and the heart vertical in position and often small.

HEART PULSATIONS The individual roentgenologist develops his own criteria for the normality of the cardiac pulsations as the subject is rotated into the oblique and lateral positions behind the fluoroscope screen. Such observations are highly subjective. In general, the larger hearts of middle and older life show less pulsation than the smaller hearts of early life. The roentgen kymograph gives a photographic record of the amplitude and direction of the pulsations of the various points on the heart borders. It is subject to error, however, and serves mainly to furnish additional information in certain instances of myocardial infarction and pericardial constriction. In the former condition, it has not the specific value of the electrocardiograph.

CALCIFICATION OF VALVES AND VALVE RINGS In demonstrating calcific deposits in the mitral and aortic valves, the x-ray may supply useful additional data. This information rarely surpasses in value the data derived from clinical examination, although routine fluoroscopic search in aged individuals might occasionally discover a calcific aortic valve missed by the clinician. Calcification of the annulus fibrosus of the mitral valve shown by roentgen examination may be taken as evidence of abnormal cardiovascular aging, but the condition does not appear to alter significantly the mechanism or functional ability of the heart and is, therefore, largely gratuitous information.

CALCIFICATION OF THE CORONARY ARTERIES This condition also may be visualized by x-rays at times, and would appear to be the clearest evidence available of abnormal degeneration of the coronary arteries. Clinical correlation has shown a disappointing lack of agreement between the x-ray finding and the cardiac dysfunction, however. Calcific coronary arteries may, in fact, have wide lumens and the patients seem, usually, to be free from anginal pain or congestive failure. In fact, Paterson¹¹ suggests that in patients with coronary disease, dietary efforts be made to produce calcification in coronary arteries to decrease the liability to occlusion by intimal hemorrhage. This is a field worthy of further investigation.

CALCIFICATION OF THE PERICARDIUM In the absence of the clinical syndrome of constrictive pericarditis, the demonstration of calcium in the pericardium is of no significance. Such a calcific deposit, it is true, is not to be expected in normal aging, but it need not shorten the subject's life. In one instance at the Massachusetts General Hospital, the whole posterior surface of the heart was encased in dense, very thick, bony-hard calcium but the patient, a man of sixty-four, worked as a carpenter up to his death from apoplexy.

THE AORTA In the absence of aneurysm, hypertensive dilatation, or senile ectasia of the aorta, the x-ray findings in the aorta tell little about the

increases about 20 per cent between the ages of fifteen and fifty, but great individual variations occur. The pulmonary artery also increases in size, but at about the age of forty-five, the aorta begins to exceed the pulmonary artery in diameter. Tortuosity, alone, gives a false picture of increased pulsa-

tion which should not be confused with aortic dilatation or aneurysm. It should be kept in mind, however, that aneurysm of the aorta on a purely arteriosclerotic basis in the aged may attain large size without serious symptoms, but eventually result in death by rupture.¹² The x ray demonstration of calcium in the aorta either in small meniscus plaques in the arch, or as heavy deposits along its entire length, tells us nothing of the effectiveness of the circulation (see p 425). Interestingly, McCay's¹³ experiments in longevity in rats have shown that those animals lived longest who developed slowly on diets balanced as to food factors, but low in calories, and that these animals had a much higher incidence of calcification of the aorta than those who were allowed to fatten at will on high quantity diets. Calcification of the aorta was, therefore, not a limiting factor in longevity.

NORMAL ELECTROCARDIOGRAM AT DIFFERENT AGES

No satisfactory studies have yet been made of the range of the normal electrocardiogram at all ages. Certain general observations may be made, however, about the changes in configuration with age, and the experience with the electrocardiographic pattern in various decades.^{14 15 16 17 18 19}

Electrocardiogram in Infancy and Childhood The infant at birth shows a preponderance of right sided electrical effect as indicated by right axis deviation in the electrocardiogram with exaggerated S wave in lead I, but with rather low amplitude of the QRS. This is evidence of the relatively large right ventricle existent at this time. The P waves may be variable in shape and amplitude, tending to be prominent, and the P-R interval short. The T wave in the chest lead in the positions from CF₂ to CF₄ may be inverted. Depending upon the growth factors of the heart and of the chest, with the increasing preponderance of left ventricular effects, the axis becomes "normal" in adolescence, and the T wave in the chest leads becomes upright. At no definite age period may this be predicted, for the right axis may change to "normal" in less than a year, and the T wave inversion in the chest lead (CF₂) may remain until the age of twenty, although it usually becomes upright by the age of ten. An inverted or diphasic T, and depressed S T interval, are also rarely seen normally in CF₄ in infancy and early childhood and notched T waves in chest leads are common at this age period.²⁰

The P-R interval increases from 0.12 second in infancy to an upper limit of 0.18 second in childhood, and the QRS from 0.06 second to 0.08 second. Savilahti¹⁷ found that up to the age of sixteen years the QRS becomes normally progressively wider according to the formula, $QRS \text{ time} = 5 + \frac{\text{age}}{4}$ (0.01 sec).

The Normal Electrocardiogram in Adult Life Detailed consideration of the normal electrocardiogram in all its aspects would require a complete chapter. It has been our practice in the Cardiac Laboratory at the Massachusetts General Hospital to consider the following criteria as approximations of normal throughout adult life.

Rate This is from 60 to 110. Below the lower rate the classification of sinus bradycardia is given, but rates from 50 to 60 with normal mechanism may occasionally be normal. White²⁷ has reported that the normal basal heart rate of champion long distance runners may be as low as 35. Above 110, the term "sinus tachycardia" is used. Its significance should be determined

in each case, as it is not a measure of abnormality of the heart, except in cases of heart failure

Rhythm Abnormalities of cardiac rhythm have been considered under the clinical examination. It is, perhaps, wise to consider all aberrations from a regular rhythm, except minor degrees of sinus arrhythmia and extrasystoles,

mature beats, auricular fibrillation and flutter, auricular and ventricular paroxysmal tachycardia—rarer conditions diagnosed by the electrocardiogram are heart block of various degrees, and nodal rhythm. Except for the latter condition, these are always signs of heart disease or abnormality. Persistent nodal rhythm or nodal tachycardia should be presumed to be evidence of heart disease. Nodal rhythm or ventricular escape as a temporary condition in the vagal hypertonia of ordinary syncope is probably not very unusual or of significance as a sign of heart disease.

P Wave In a normal record, it should be possible to find a well marked P wave in at least one of the standard leads. In lead III, the P wave is often isoelectric or inverted, and there may be a respiratory change of direction in this lead. When the P wave in leads II and III is inverted, nodal rhythm may be the cause if associated with a short P-R interval. If it changes its shape or direction from beat to beat, the term "wandering pacemaker" is used. This has much the same significance as marked sinus arrhythmia discussed previously. In leads I and II, the limits of normal amplitude of the P wave are 0.05–0.25 millivolt. Notching of the P wave is probably normal at all ages. Inversion of the P wave in lead I or lead II probably indicates an abnormal auricular muscle or abnormal position of the pacemaker.

P-R Interval The length of the P-R interval is related somewhat to rate. Lengthening with increase in rate suggests some slight fault in conduction. It should be measured in the lead showing the longest interval, but it must be observed whether or not the interval is definitely shorter in the other two leads, since a balance of electrical effect in the early part of the QRS may, in this lead, result in an isoelectric interval preceding the QRS giving a false lengthening to the P-R interval. The upper limit of the P-R interval to the age of fourteen is 0.16 second. In adolescence, 0.18 second may be allowed, in adult life, 0.2 second. Occasionally P-R intervals of 0.21 second or 0.22 second are seen in apparently normal young males with bradycardia.

QRS Duration In childhood, the average QRS duration time is 0.08 second, with a normal limit of 0.09 second.¹⁷ In adults, the average is 0.09 second with the upper limit 0.12 second. It should be measured in the axial lead showing the greatest duration. Slurring near the baseline is normal, as is notching and splitting of the QRS in lead III. Notching is not normal in leads I and II.

QRS Amplitude No definite limit of normality of amplitude of the QRS can be stated. When the deviation from the baseline does not exceed 0.5 millivolts in either direction in all three limb leads, the tracing is classified as of low voltage. The upper limit by the 'Nomenclature and Criteria for the Diagnosis of Diseases of the Heart'¹⁸ is 2.0 millivolts.

Deep Q₃ The "Nomenclature and Criteria for Diagnosis of Diseases of the Heart"¹⁸ states as follows concerning the Q wave: "A Q wave is considered

in relation to the largest QRS deflection in the three leads. If Q_1 is greater than 15 per cent of this deflection, or Q_2 is greater than 20 per cent, or Q_3 is greater than 25 per cent, it is regarded as deep. These criteria do not hold in electrocardiograms showing a high voltage of the QRS group, or for Leads II or III if there is right axis deviation. A deep Q_3 is not necessarily abnormal. In a series of very carefully followed employees of the Metropolitan Life Insurance Company,²¹ aged forty years or over, without any evidence of heart disease, it was present in 6 per cent of their electrocardiograms. Its probable abnormality is much increased when associated with an inverted T_3 , and a Q in lead II. If T_2 also is flat, and almost certainly if inverted, this combination of signs is evidence of myocardial infarction of the basal type. Inversion of T_2 and T_3 in the absence of well marked Q waves is not always diagnostic of myocardial infarct. Other conditions, such as are listed later, specifically pulmonary embolism, high diaphragm, emotional influences, and alkalosis from hyperventilation, should be ruled out.

Axis Deviation. As growth occurs, the right axis deviation of childhood becomes the normal axis of adult life, and the slight left axis of later adult life and old age. This shift occurs although the influence of a high diaphragm with transverse position of the heart and the influence of hypertension are eliminated. If the Einthoven triangle method is used to measure the degree of left axis deviation, a shift above the horizontal of 0 degrees is considered abnormal, while by the index method, a value of more than plus 20 is abnormal. Remarkable shifts to a normal axis in some cases may be produced by full inspiration, and are reassuring evidence against the diagnosis of an abnormal left ventricular strain.

Right axis deviation may be normal in tall, thin individuals with vertical hearts. Its occurrence in persons without this body build is abnormal.

S-T Segment Changes. An elevation or depression of the origin of the S-T segment of more than 0.1 millivolt is classed as abnormal. Sagging of the S-T segment or upward curving is abnormal.

T Wave. The T wave may normally be flat, inverted, or diphasic in lead III. Occasionally, it may be low or inverted in lead II or in leads II and III in normal individuals, but should become upright on change of position to recumbency, or on change of the position of the diaphragm with respiration. The T wave is never normally flat or inverted in lead I. These alterations of the S-T and T segments are not necessarily indicative of cardiac disease, or at least are not diagnostic of coronary artery degeneration. The conditions noted in the following tabulation may produce such changes. Others undoubtedly exist. Similar effects due to sickle cell anemia and deposit of blood pigments in the heart muscle in hemochromatosis have been recently noted. King²³ states, for example, that heavy consumers of alcohol may show flat or inverted T waves in any or all leads without relationship to dietary deficiencies. All such factors should be eliminated, if possible, before attributing nonspecific electrocardiographic changes to degenerative coronary disease with myocardial ischemia.

Precordial Leads. Data on the precordial leads are as yet insufficient to define the limits of normality in various derivations. In general, in the adult,

from the right

CHAPTER 25

DISTURBANCES OF CONDUCTION AND RHYTHM

LOUIS B. LAPLACE

THE HEART RATE

THE average heart rate is said to become somewhat slower in old age, having remained relatively constant during middle life¹ The change is attributable to a number of contributory causes, including decrease in the sensitivity of the heart to vasomotor influences, lowering of the body metabolism, and reduction of physical activity Slowing of the heart during senescence has not, however, been consistently demonstrated in the reported studies of the subject^{2, 3} and is probably so slight as to have little or no clinical importance

The reaction of the heart rate under various circumstances is modified by advancing age in a more or less consistent manner The effect of nervous influence is diminished, the heart accelerates less in response to atropine⁴ and slows less in response to carotid sinus pressure⁵ than it does in earlier life A relatively faster rate is induced by a standard exercise test,⁶ but the maximum heart rate attainable by physical exertion is decreased

The adaptability of both the heart and peripheral vascular system to fast and slow rates is substantially diminished in old age Marked bradycardia is more likely to induce syncopal manifestations and slowing of the rate during sleep has been found to be a potential cause of nocturnal paroxysmal dyspnea⁶ This is presumably due to progressive limitation of the capacity of the heart to increase its output per beat Tachycardia is likewise poorly tolerated by the aged It increases the oxygen consumption of the myocardium to an extent which tends to exceed the ability of sclerotic coronary arteries to supply an adequate blood flow The consequent state of relative coronary insufficiency is commonly a cause of anginal pain or even acute myocardial infarction (See Chapter 27) Peripheral circulatory insufficiency is also more easily induced by tachycardia in old age because the reduction in cardiac output is less effectively compensated for owing to senescent changes in the vascular system (See Chapter 30) Under these circumstances it is apparent that control of the heart rate in aged patients is of relatively greater therapeutic importance than it is in earlier life

ARRHYTHMIAS

The normal rhythm of the heart is produced by the periodic discharge of an electrical impulse from the sinoauricular node (S-A node) The impulse is the stimulus which initiates myocardial contraction The activity of the S-A node is normally modified by physiologic reflex and chemical influences which cause increase, decrease, or phasic irregularity of the heart rate according to the requirements of the circulation A rapid rhythm with a rate of 100 per minute or more is known as sinus tachycardia A slow rhythm of 60 per minute or less is known as sinus bradycardia A phasic variation of

rate is known as sinus arrhythmia. The sinus rhythms are usually physiologic but may also reflect both cardiac and extracardiac abnormalities, especially toxic and metabolic disorders. In old age, the sensitivity of the S-A node tends to decrease and pronounced variations of sinus rhythm are less often the result of the physiologic nervous influences to which they are usually attributable in young subjects.

Abnormal cardiac mechanisms occurring outside of the S-A node may cause a variety of arrhythmias. The abnormal mechanism may be located in the auricles, auriculoventricular node (A-V node), or ventricles. Arrhythmias arising in the A-V node are often indistinguishable from those of auricular origin so that the term supraventricular is used to include both sites of origin. The arrhythmias of ectopic origin may be grouped into the following predominant types: premature systole, nodal rhythm, paroxysmal tachycardia, flutter, and fibrillation.

Premature systole consists of an isolated beat of ectopic origin. Two such beats occurring in succession are also considered to be premature systoles but a succession of three or more is regarded as paroxysmal tachycardia. Premature systoles may occur sporadically, in coupled rhythm (pulsus bigeminus), or in a rhythm quite independent of the dominant cardiac rhythm. Their incidence is increased with age and this form of ectopic beat undoubtedly occurs at some time in all persons over sixty years. Premature systoles are usually less distressing to elderly patients than they are to younger subjects and ordinarily produce no subjective symptoms whatever.⁷

Nodal rhythm is a form of abnormal cardiac mechanism in which the dominant cardiac pacemaker is located in the A-V node. The cardiac rate may be normal or relatively slow or it may be extremely rapid, having the general characteristics of paroxysmal tachycardia.

Paroxysmal tachycardia is a rapid succession of three or more ectopic beats. The rate ranges from 90 to 240 or higher but is generally between 140 and 180. The onset and termination of the paroxysm are usually abrupt. Individual attacks vary in duration from a few seconds to several months. In elderly subjects, the danger of paroxysmal tachycardia is proportional to the rapidity of the rate, the duration of the attack, and the functional state of the heart. Thus rates under 150 may be fairly well tolerated, temporarily at least, causing only weakness and discomfort, but rates over 150 generally lead to pulmonary edema and other manifestations of heart failure in a relatively short time. Ventricular tachycardia is a far more serious condition than the auricular form, being commonly associated with an advanced degree of myocardial disease.

Flutter consists of rapid rhythmic contractions of the auricles or ventricles in which, unlike paroxysmal tachycardia, neither systole nor diastole involves the entirety of the affected chamber at the same time, one part being in systole while the other is in diastole. In auricular flutter, the auricles beat rhythmically at a rate commonly between 200 and 350. The ventricles rarely respond to every auricular beat but rather to every second, fourth, or fifth beat. As in paroxysmal tachycardia, the chief danger to elderly subjects is the occurrence of a prolonged acceleration of the ventricular rate tending to produce myocardial exhaustion. In ventricular flutter, the rate averages 250. This condition is usually a prelude to fatal ventricular fibrillation and rapidly induces syncope.

Fibrillation is essentially similar to flutter except that the contractions are arrhythmic, multiple scattered areas of the affected chamber being in systole while the others are in diastole. In auricular fibrillation, the rate of auricular contraction averages 250 to 400. The ventricles respond at a rate determined by the conductivity of the A-V conduction system. The ventricular rhythm is totally irregular except when high degrees of A-V block are present. As with other arrhythmias, elderly subjects tolerate auricular fibrillation poorly when the ventricular rate is excessively rapid. Fibrillation of the ventricles is incompatible with life since it involves complete loss of propulsive power of the heart.

The genesis of these arrhythmias of ectopic origin is still uncertain in many respects. Three types of mechanism have been postulated: sporadic pacemakers, a re-entry phenomenon, and parasystole. The existence of a sporadic pacemaker is suggested by the occurrence of isolated and apparently independent premature systoles. Presumably such a pacemaker is normally suppressed by the more rapid action of the S-A node. It is able to activate the heart, however, when its rhythm is accelerated and temporary local block prevents the normal impulses from reaching and suppressing it. The theory of re-entry is suggested by the regular recurrence of premature systoles at a fixed interval following the dominant beats, especially when the dominant rhythm is irregular. If a certain area of the heart has a sufficiently prolonged refractory period, it will fail to be activated at the same time as the surrounding tissue. As electrical activity is subsiding elsewhere, however, the refractory area becomes responsive, undergoes a delayed activation and in turn reactivates the remainder of the heart.

The theory of parasystole postulates the existence of active ectopic pacemakers in the heart. These pacemakers usually dominate areas which are too small for electrocardiographic detection and are surrounded by areas of unidirectional block due to local prolongation of the refractory period. Changes in the surrounding block may permit the occurrence of a parasystolic rhythm. Such a rhythm is suggested when the intervals between premature beats from the same focus are equal to each other or are multiples having a common denominator.

Both re-entry and parasystole are logical explanations of the genesis not only of premature systoles but also of paroxysmal tachycardia, flutter, and fibrillation. These rhythms might originate from one or more ectopic pacemakers having a rapid rate of discharge or could be interpreted as being caused by repeated re-entry from one or several points. It is possible in fact that the activation of ectopic pacemakers and the phenomenon of re-entry may be part of the same fundamental process of which the different arrhythmias are varied but basically related manifestations.

A clinical diagnosis of the particular type of arrhythmia usually can be made by the methods of physical examination. The diagnostic criteria involved are outlined below. Often, however, an accurate diagnosis can be made only by electrocardiography and occasionally even this procedure may fail to reveal the precise mechanism involved, especially when the rhythm is very rapid and some of the complexes are obscured.

Sinus Tachycardia. Simple (sinus) tachycardia is more significant in old age than in youth and a resting rate of over 100 per minute is usually an indication for careful study.⁸ The common causes are extracardiac, such as

febrile disease and various toxic states, especially hyperthyroidism. Simple tachycardia may, of course, accompany heart disease but in the majority of elderly patients who have cardiac impairment, the rate is not particularly fast.

The clinical *differentiation* of simple tachycardia from other rapid regular rhythms is based on the fact that the rate can be altered by change of posture and that *gradual slowing and acceleration are produced by the application and withdrawal of digital pressure on the carotid sinus*.

The *treatment* of sinus tachycardia is directed against the underlying cause. Digitalis should not be used unless congestive failure is present as its effect on the sinus mechanism is negligible and it is often definitely harmful to the senile heart.

Sinus Bradycardia. Simple (sinus) bradycardia is common among old people who are in relatively good health.⁹ In such persons it is probably attributable to decreasing sensitivity of the sinus node and to decrease in body metabolism. Pathologic causes include hypothyroidism, disorders of the nervous system, reflexes arising from the gastrointestinal tract, and various toxic states. Rates of 50 per minute or less are likely to induce considerable inadequacy of the circulation, especially if any form of cardiovascular insufficiency is present, since the output of the senile heart under these circumstances is substantially reduced.

Simple bradycardia may be *differentiated* clinically from other slow rhythms by the fact that the rate accelerates normally (rather than abruptly as in ectopic rhythms and incomplete heart block) in response to exercise.

Treatment of simple bradycardia is unnecessary if there is no evidence that it interferes with cardiovascular efficiency. When acceleration of the rate is desired atropine may be used, although its effectiveness is relatively decreased in advanced age.⁴ Caution should attend the administration of thyroid extract because of its tendency to aggravate the effects of coronary disease.

The question occasionally arises as to whether the presence of sinus bradycardia is a contraindication to the use of digitalis. In a case of congestive heart failure, the drug should not be withheld because of a slow rate as therapeutic doses of the drug are unlikely to produce any further slowing.

Sinus Arrhythmia. Phasic irregularity of sinus rhythm is most pronounced in youth and diminishes with age, and it is characteristic of the normal senile heart that its rhythm is relatively "fixed."⁹ The presence of marked sinus arrhythmia in old age is usually pathologic. There are many disorders which tend to produce it by accentuating the influence of respiration on blood flow. Phasic arrhythmia may be a conspicuous feature of acidosis, anoxemia, and other toxic states.

Sinus arrhythmia is *differentiated* clinically from other irregular rhythms which it may resemble, such as auricular fibrillation and premature beats, by its consistent relationship to the respiratory rhythm.

Treatment of the arrhythmia is not required but attention should be directed to the underlying cause. The only importance of sinus arrhythmia in the aged is its diagnostic significance.

Auricular Premature Systoles. Premature systoles arising from an ectopic focus in the auricles are less frequent than those of ventricular origin. Their causes range from inconsequential nervous or toxic effects to organic myo-

cardial damage, so that their practical importance depends entirely on the associated clinical evidence. Although they usually reflect some abnormality of the auricles, in old people this does not necessarily imply anything more serious than senile fibrotic change. In certain cases, however, auricular premature systoles may precede and follow attacks of auricular paroxysmal tachycardia, fibrillation, or flutter, and recognition of the type of ectopic beat may clarify the diagnosis of an otherwise unexplained history of palpitation.

Auricular premature systoles are often distinguishable from those arising in the ventricles by the fact that they produce less change in the character of the first heart sound.

Treatment is not required unless the symptoms are distressing or an attempt is being made to prevent a paroxysmal arrhythmia. It is often difficult to completely abolish the ectopic beats. Any measures which improve the general hygiene may be helpful. The drug most often successful is quinidine, but digitalis may also be effective and should be tried first because of its greater convenience of administration.

Auriculoventricular Nodal Beats Ectopic beats which originate in the auriculoventricular node or common bundle are less frequent than those arising elsewhere but are more often of clinical importance. They are the type which tends to occur following prolonged diastole (ventricular escape). In elderly patients they are likely to be associated with coronary disease. The toxic effect of digitalis is a common cause.

Nodal beats can seldom be *differentiated* from other premature systoles except by graphic methods.

The *treatment* of nodal beats in cases in which digitalis toxemia is suspected consists of tentative withdrawal of the drug. In other cases the arrhythmia is often terminated by digitalis. When coronary disease is apparently responsible, drugs of the xanthine or nitrite group may be indicated.

Ventricular Premature Systoles Premature systoles arising from an ectopic focus in the ventricles (below the bifurcation of the common bundle) are the type most frequently encountered in all ages and are the least likely to be of any clinical importance. They are usually significant, however, when they occur in large numbers in a subject whose rhythm has previously been regular, or when they occur in coupled rhythm (pulsus bigeminus). Coupled rhythm is often a toxic effect of digitalis. Premature systoles occurring in sequence of two or more may be a premonitory sign of paroxysmal ventricular tachycardia. When they occur in the presence of complete heart block, their presence may have a beneficial effect by increasing the ventricular rate. The ventricular origin of premature systoles may be detected by the difference in character of the first heart sound which lacks the element contributed by auricular contraction.

Treatment of ventricular premature systoles is seldom indicated in the aged except for special reasons. Sedative drugs have little value in the absence of a conspicuous psychogenic factor. Gastrointestinal disorders are a common cause, the correction of which may be effective in abolishing the arrhythmia. (See Chapters 33 and 34.) If there is evidence of cardiac insufficiency, the premature beats may disappear following the administration of digitalis, but if digitalis has previously been given in large doses it should be withheld. When ventricular premature systoles occur in the course of acute myocardial

infarction it is good practice to give quinidine in order to prevent a possible attack of paroxysmal ventricular tachycardia. Quinidine is also indicated when premature beats accompany attacks of angina pectoris.

Auricular Paroxysmal Tachycardia Paroxysmal tachycardia arising from an ectopic focus in the auricles occurs less often in advanced age than in middle life. It is much less likely to be associated with serious heart disease than either the nodal or ventricular types, and in some cases no other clinical abnormality is apparent. It also tends to be of shorter duration and will almost always terminate spontaneously. It is poorly tolerated in the aged however, because of the tendency to acute coronary insufficiency and myocardial exhaustion by the fast rate. The attacks are often accompanied by precordial pain and should be considered as a possible cause of otherwise unexplained episodes of spontaneous angina.

Auricular paroxysmal tachycardia is distinguished clinically by the regular rhythm and the rapid rate which is most commonly between 140 and 180 per minute. The diagnosis can be established if the attack is terminated by vagal excitation.

Treatment of the attack consists in the use of various measures which produce vagal excitation. These include digital pressure over the right carotid sinus, holding the breath, drinking ice water, bending over a chair, or inducing nausea by tickling the back of the throat. Pressure on the eyeballs is widely recommended but seems scarcely an appropriate treatment for an aged patient. The most effective remedy is the injection of acetyl beta methyl choline (mecholyl) which is a potent vagal stimulant. The side effects of this drug are sufficiently dangerous, however, that it should not be used in old people unless simpler methods have failed. Syrup of ipecac is also an effective vagal stimulant, its administration is considerably safer than the use of acetyl beta methylcholine. As a rule, digitalis is the drug of choice. Quinidine and quinine, which act directly on the auricular myocardium, are likewise capable of terminating an attack, and in emergency may be given intravenously. Other therapeutic measures which have been used successfully include morphine, intravenous calcium, magnesium sulfate, and papaverine.

Prevention of the attacks is often accomplished by general measures, such as elimination of psychogenic causes, control of intercurrent disease and attention to gastrointestinal function. The most satisfactory drug for prophylactic use is digitalis, owing to its safety and convenience of administration. Quinidine should be used if digitalis therapy fails.

Auriculoventricular Nodal Rhythm and Tachycardia Nodal rhythms of all types may be produced by coronary disease so that their incidence increases with advancing age. Nodal tachycardia is often found to be a toxic effect of digitalis. The tachycardia is a serious handicap to the senile cardiovascular system but the slower rhythm may cause no apparent ill effect.

Nodal rhythm is seldom recognized clinically when the rate is not accelerated. When the rate is rapid, electrocardiographic study is almost always required to differentiate the condition with certainty from other forms of tachycardia.

Treatment of the underlying cause when this is apparent, is always indicated. Since the toxic effect of digitalis may be responsible for both the slow and rapid rhythms, this drug should be withdrawn in any case in which

it was being administered when the arrhythmia started. Digitalis therapy, however, is the most effective method of terminating nodal tachycardia in patients to whom it has not previously been given. Quinidine may be used alternatively. No treatment is required to counteract the slow rhythm unless the bradycardia is excessive, in which case atropine is sometimes helpful, especially if vagal influence appears to be a contributory factor.

Ventricular Paroxysmal Tachycardia Paroxysmal tachycardia arising from an ectopic focus in the ventricles exhibits a rate which averages between 150 and 250 per minute. It is usually associated with severe coronary disease, especially acute myocardial infarction. It is always a very grave complication, not only because of the strain which it imposes on a seriously damaged heart, but also because of its tendency to merge into fatal ventricular fibrillation. Among the aged, therefore, it is encountered most often as a terminal episode. Every attack of ventricular tachycardia should be treated as an emergency. Recovery occasionally ensues, however, even after the arrhythmia has persisted for several days.

Ventricular tachycardia is often distinguishable from other rapid rhythms by the fact that the rate tends to vary slightly from minute to minute. There is also a slight change in the character of the first heart sound every few seconds as the auricular and ventricular contractions approach and regress from their normal time relationship. Finally, the heart rate is not altered by pressure on the carotid sinus.

Treatment consists in the oral administration of quinidine sulfate or the intravenous or intramuscular injection of quinidine or quinine in increasing dosage until the tachycardia is stopped. When given intravenously, these drugs should be diluted in 50 cc. of saline solution to prevent convulsive seizures. Digitalis and mechohyl are contraindicated. If quinidine therapy fails, intravenous administration of morphine, calcium, magnesium sulfate, papaverine, or procaine should be tried.

Auricular Fibrillation Auricular fibrillation is very common in the aged. It has been found in 3 per cent of persons over seventy years whose hearts appeared to be otherwise normal on clinical examination⁸ and in 17 per cent of unselected hospital patients of the same age.¹⁰ In later life, the arrhythmia is almost always chronic and the paroxysmal form is rarely encountered except during the acute stage of myocardial infarction. It appears that once auricular fibrillation becomes established in an aged patient, the underlying senescent changes in the auricles are sufficient to perpetuate it as a permanent rhythm.

Although commonly benign, auricular fibrillation does occur, as in earlier years, as a result of active or impending congestive heart failure which it tends to aggravate. On the other hand, its onset may be the precipitating cause of congestive failure especially if the ventricular rate is rapid. Among elderly persons, however, the ventricular rate tends to be slower than in youth due to the impairment of conduction which accompanies senescence, and the patient may be completely unaware of the existence of the arrhythmia. In those cases in which the ventricular rate is very fast, auricular fibrillation, like other forms of tachycardia, is poorly tolerated.

The clinical *diagnosis* of auricular fibrillation is based on the complete irregularity of the ventricular rhythm. When the ventricular rate is either

very fast or very slow, the irregularity is less apparent and may be difficult to recognize. In the presence of complete heart block, the ventricular rate is regular, but in such cases the arrhythmia of the auricles is of little consequence.

The *treatment* of auricular fibrillation in the aged should seldom involve an attempt to abolish the arrhythmia once it has become established. There is no indication for quinidine therefore, except when auricular fibrillation occurs in the course of acute myocardial infarction. Even in such cases, however, the arrhythmia usually terminates spontaneously in a short time so that treatment is seldom necessary unless the ventricular rate is fast and precipitates heart failure.

The usual treatment of auricular fibrillation is the administration of digitalis in a maintenance dose sufficient to keep the ventricular rate within the desired limits. In the average case this consists of $1\frac{1}{2}$ grains of powdered leaf (U S P XIII) daily. Treatment is not necessarily required, however, if the ventricular rate at rest is 85 per minute or less and if it does not accelerate excessively during mild physical activity.

Auricular Flutter. Flutter of the auricles is much less common than fibrillation. Among the aged, the ventricular rate tends to be slower than in earlier life due to impairment of conductivity. When the ventricular rate is slow, the arrhythmia has no significant ill effect, the danger of auricular flutter lies in the fact that a change to a 2:1 or 1:1 response will precipitate an exhausting tachycardia.

Auricular flutter is easily overlooked if the ventricular rate is within normal limits. When the ventricular rate is fast, the arrhythmia resembles paroxysmal tachycardia. It may be identified clinically if pressure on the carotid sinus changes the auriculoventricular response and causes the ventricles to beat irregularly or at a slower but constant rate, with return to the faster rate when the reaction ceases.

Treatment consists in the administration of sufficient digitalis to convert the flutter into fibrillation and produce adequate slowing of the ventricles. When the arrhythmia is of recent onset, this procedure is often followed by reversion to normal rhythm. Among elderly persons, termination of the arrhythmia by quinidine therapy is rarely indicated except when the flutter is paroxysmal in type. Paroxysms of auricular flutter are not uncommon during the acute stage of myocardial infarction, but these attacks usually terminate spontaneously. Quinidine may be given during the attack to accelerate the change and afterward to prevent recurrence. In the rare cases of 1 to 1 response in which the ventricular rate is extremely fast, digitalis constitutes an emergency measure and intravenous administration is often desirable.

Ventricular Fibrillation. With the onset of this arrhythmia the circulation comes to a standstill. In rare instances, ventricular fibrillation occurs in brief paroxysms and produces Stokes-Adams attacks, but in the vast majority of cases, it is simply a terminal event and life endures only a few minutes after the onset.

Treatment is entirely prophylactic, for there is no practical method whereby the arrhythmia can be terminated except during surgical operations when the heart is directly accessible. The intracardiac administration of adrenalin is considered more likely to precipitate or perpetuate ventricular fibrillation than to abolish it and should therefore be abandoned as a routine.

procedure in cases of acute heart failure. There is evidence that the mortality due to ventricular fibrillation in the course of acute myocardial infarction is lowered by the routine use of quinidine.

CONDUCTION DEFECTS

The pathway over which impulses from the auricles must travel in order to reach and activate the ventricular myocardium includes the A-V node, the common bundle, the branch bundles, and the Purkinje fibers. Pathologic changes alter the conductivity of these structures and cause the transmission of impulses to be delayed or completely blocked. Involvement of the A-V node or common bundle produces various degrees of heart block. Involvement of one of the branch bundles produces intraventricular block. Conduction defects are found with increasing frequency in old age since their commonest cause is fibrotic change associated with coronary sclerosis.

Auriculoventricular Block. Incomplete heart block of minimal degree consists of simple delay in auriculoventricular conduction as indicated by prolongation of the P-R intervals of the electrocardiogram. The duration of the average normal P-R interval is 0.16 second. A slight increase in conduction time tends to occur with advanced age and P-R intervals of 0.20 second, which in youth are usually the result of pathologic change, are considered to be within normal limits in later life. P-R intervals of longer duration may occur in old people without other evidence of heart disease, but are generally attributable to coronary disease involving the area of the A-V node or common bundle. Such lesions may be relatively benign or may be part of more extensive and serious myocardial damage.

Prolonged conduction time has no significant ill effect on heart function. It is important chiefly as evidence of underlying myocardial disease and because further progress of the lesion may result in higher grades of block which usually produce discomfort or even syncope. The diagnosis is often suggested by the presence of a presystolic gallop sound but is seldom established without graphic recording.

Incomplete heart block of higher grade involves failure of some of the impulses to traverse the conduction system. This is manifested clinically as a "dropped beat." Dropped beats may occur occasionally or in more or less regular sequence. The effect on circulatory function is variable and, as a rule, depends upon the frequency with which the dropped beats occur. In some cases there are no symptoms but in others the condition may be very distressing and may even cause Stokes-Adams attacks. Such attacks are most likely to occur when the ventricular rate is suddenly reduced to one-half or one-third by an increase in the number of dropped beats. When any form of heart block occurs in the course of acute myocardial infarction, the prog-

sound of a normal beat should be heard.

Complete heart block consists of an obstruction which prevents any of the auricular impulses from activating the ventricles and renders the auricular and ventricular rhythms entirely independent of one another. The

ventricles contract in response to a pacemaker in the common bundle which discharges rhythmically at rates, as a rule, between 30 and 40 per minute. Such slow rates are seldom well tolerated by old people, especially on exertion, because of the relative inability of the heart to increase its output per beat sufficiently to maintain adequate blood flow. Dizziness and various mental symptoms are therefore common. Actual Stokes-Adams attacks are usually caused by failure of the ventricular pacemaker to discharge for 5 seconds or more during which time ventricular systole is arrested and the circulation comes to a standstill.

Complete heart block is recognized by the slow rate which is not significantly accelerated by physical activity. The diagnosis can be made with reasonable certainty when the auricular beats are audible, or when it is possible to detect the phasic variation in intensity of the first heart sound due to change in time relationship of the auricular and ventricular contractions.

Treatment of prolonged auriculoventricular conduction is not required although appropriate measures may be indicated for the underlying myocardial disease. Prolonged conduction is usually an indication for caution in the use of large doses of digitalis which are potentially capable of increasing the obstruction and inducing dropped beats. When digitalis is a cause of defective conduction, it should, of course, be withdrawn.

Incomplete block with dropped beats may progress to complete block as the obstructive process becomes more effective, but in the majority of instances regular rhythm returns either spontaneously or in response to therapy. Digitalis should be withheld unless there is a substantial degree of congestive failure. The auricular rate should be kept as slow as possible so as to permit the maximum time for recovery in the conduction system between beats. This may require temporary confinement to bed. Appropriate drugs include coronary dilators and myocardial stimulants. Atropine may benefit or aggravate the condition, depending upon the effect of its influence in accelerating the auricles as compared with blocking vagal inhibition of the auriculoventricular node.

The treatment of complete block is very unsatisfactory. It is desirable to reestablish coordinated rhythm if this is possible. The same procedures are employed as in the treatment of incomplete block, but they are usually ineffective, except in cases where the block is due to an acute process which tends to subside. When complete block is permanent, the object of therapy is to increase the ventricular rate and prevent the periods of cardiac standstill which produce Stokes Adams attacks. A significant degree of acceleration is very difficult to obtain. Under the circumstances, the occurrence of ventricular extrasystoles may be actually beneficial since their presence involves an increase in the ventricular rate. Drugs of the xanthine and ephedrine groups as well as various myocardial stimulants, may be tried but if, as often happens, they increase the work of the heart without proportionately increasing

ventricular pacemaker. Only in cases where it seems possible that normal rhythm may be reestablished is it advisable to withhold the drug tentatively. Otherwise digitalis may, and usually should, be given if there is any evidence myocardial insufficiency.

Intraventricular Block. Defective transmission of impulses through one of the branch bundles or their subdivisions produces asynchronous contraction of the two ventricles. This does not appear to cause any significant handicap to heart function. Conduction defects of this type may be produced by cardiac lesions which vary in extent from a large infarct, which is incompatible with life, to minute areas of fibrosis which have little or no practical importance. The clinical significance which is attached to the finding of a bundle branch block or a lesser degree of intraventricular block depends very largely therefore on the degree of myocardial damage which is associated with it.

Senescent changes in the heart lead to an increasing incidence of various degrees of intraventricular block in the later years of life. As might be expected from the nature of their pathogenesis, they are common among old people who have no clinical evidence of heart disease and have been found in as high as 24 per cent of such a series.⁶ They are detected only by means of electrocardiographic recording.

No treatment is indicated unless required by the underlying myocardial disease. Digitalis is said to aggravate the degree of block and should be given with caution.

THE ELECTROCARDIOGRAM IN OLD AGE

The electrocardiogram may be normal even in very aged subjects (See Chapter 24.) It is said to be unaltered by the usual senile changes in the heart—brown pigmentation, increase in connective tissue and fat deposits in the conduction system.¹¹ Although it may be similar in all respects to that of a healthy young subject,¹² the electrocardiogram more commonly tends to depart from its standard pattern in a manner considered characteristic of senescence (See p. 367.) The electrical axis shifts toward the left, the P-R and QRS intervals lengthen and the voltage of the major deflections decreases.¹ These alterations may not be actually "normal," but the underlying changes which they represent are, as a rule, so benign that they have relatively little clinical importance.

Myocardial damage often fails to alter the electrocardiogram and this accounts for many instances of normal records among the aged. Coronary disease, which is so prevalent in later life, is especially prone to be unaccompanied by any commensurate degree of graphic evidence. The extent to which cardiac changes may progress in the presence of a normal electrocardiogram is illustrated in Figure 71. Under the circumstances it is clearly fallacious to evaluate the condition of the heart on the basis of an electrocardiogram alone and without correlation of the clinical data.

The Electrocardiogram in the Absence of Clinical Evidence of Heart Disease. The extent, or even the presence, of myocardial disease in the aged is often impossible to determine with accuracy. It has been stated that "a normal heart is practically never observed at necropsy in a person who has passed the age of seventy-five years."¹³ Nevertheless, 45 per cent of the patients of this age who attended the Mayo Clinic were found to exhibit no significant abnormality of the heart on clinical examination.¹⁴ Among such persons changes in the electrocardiogram are commonly the result of "silent" coronary disease.

Normal Electrocardiograms. There is a wide disparity in the incidence of normal electrocardiograms as reported by different observers. In one

series only 15 per cent were normal,⁸ whereas in another all of the records were considered to be without significant abnormality.¹⁴ In two additional series the reported incidences of normal electrocardiograms were 58 and 74 per cent.^{7, 15} Such difference of opinion presumably arises from lack of uniformity in the criteria employed. It indicates clearly some of the difficulties involved in determining what constitutes a normal heart in old age.

Arrhythmias Premature systoles constitute the most common of the arrhythmias and have been found in as many as 34.4 per cent of electro-

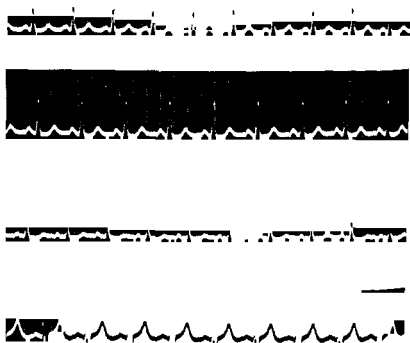


Fig. 71 Normal electrocardiogram in the presence of advanced heart disease. Subject male, age sixty-five years. Although the electrocardiogram is essentially normal, physical examination revealed huge cardiac enlargement and severe congestive failure due to hypertensive and arteriosclerotic heart disease. This illustrates very strikingly the fact that a complete cardiac diagnosis should never be based on the electrocardiogram without correlation of the clinical evidence, especially in later life.

cardiograms.¹⁴ Since they are not continually present, however, they often fail to be recorded and it is probable that they occur at some time in practically all persons more than sixty years of age. Ventricular premature systoles are about twice as frequent in later life as those arising from the auricles.¹⁰ Auricular fibrillation is occasionally found in a heart which appears otherwise normal and has been reported in 3 per cent of such cases.⁸ Auricular flutter and various forms of paroxysmal tachycardia have not been reported in elderly patients without other associated evidence of heart disease.

Conduction Defects Auriculoventricular conduction tends to be slightly longer in old age than in youth and P-R intervals of 0.20 second are within normal limits. P-R intervals slightly in excess of 0.20 second have been reported to occur in as many as 40 per cent of cases⁸ but 1.7 to 10 per cent is the range more often quoted for apparently normal subjects.^{3, 7} Dropped beats and complete A-V block are not found in the tracings from a normal heart. Incomplete intraventricular block occurs in approximately 4 per cent of cases.^{3, 8, 14, 15} Bundle branch block (QRS intervals of 0.14 second or

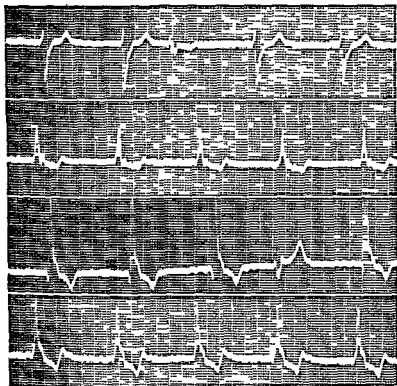


Fig. 72 Numerous electrocardiographic abnormalities in a patient who had surprisingly good cardiovascular function. Subject male aged sixty-eight years. The electrocardiogram revealed auricular fibrillation, defective auriculoventricular conduction (heart block), ventricular premature systoles, and a right bundle branch block. In spite of these arrhythmias and conduction defects which were associated with coronary disease the patient survived an extensive laparotomy, required no cardiac medication and had no manifestations of cardiac insufficiency except dyspnea on climbing stairs.

more) has been reported in 20 per cent of one series⁸ but 7 per cent seems closer to the average incidence.^{10, 15}

Axis Deviation. Left axis deviation is the most common change in the electrocardiogram in later life, its incidence being somewhat more than 50 per cent.^{3, 8, 9, 10} In the majority of cases it is at least partly due to a transverse position of the heart and therefore represents a physiologic rather than a pathologic effect (see p. 364). Right axis deviation in the aged is said to be rare in the absence of a definitely pathologic cause.⁸

Q Wave. A prominent Q wave has been reported to occur in lead I in

1 per cent and in lead III in 3 per cent of cases.⁸ A prominent Q wave in lead I is almost always pathologic, but in lead III is not necessarily so.

S-T Interval S-T interval deviation in one or more leads has been reported to occur in 0.3 to 7 per cent of cases.^{3, 15} Its clinical significance is uncertain in the absence of acute myocardial injury or drug (digitalis) effect.

T Wave Changes in the T wave in lead I alone have been reported in 19 per cent of cases,⁸ but definite inversion has been found in only 3 per cent.¹⁵ Similar changes in the T waves of both leads I and II are less frequent. An inverted T wave in lead I is practically always the result of pathologic changes in the heart, but flattening without definite inversion cannot be regarded as more than a suspicious sign. Inversion of the T wave in lead III is not considered abnormal unless there is other evidence to this effect.

Low Voltage Decrease in amplitude of the major deflections is less commonly a sign of heart disease in old age than it is in earlier life. Its reported incidence in different series varies between 13 and 37 per cent.⁸

The Electrocardiogram in the Presence of Clinical Evidence of Heart Disease. The incidence of abnormalities of the electrocardiogram is considerably higher among old people who show clinical evidence of heart disease than among those who do not. The types of changes are similar except for the occurrence of additional arrhythmias and conduction defects together with patterns which are characteristic of acute myocardial injury, such as infarction and pericarditis. The incidence of normal electrocardiograms, in spite of demonstrable heart disease, in this group is approximately 10 per cent in the eighth decade and 3 per cent in the ninth decade.¹⁰

Premature systoles have been reported to occur with about the same frequency as in persons whose hearts are apparently normal.^{14 16} Auricular fibrillation is much more common and occurs in approximately 18 per cent.^{10 14} Auricular flutter and complete heart block have been found in 1 per cent of cases.^{10 14}

The incidence of left axis deviation is not significantly increased, but right axis deviation has been found in approximately 1 per cent of cases.^{3 10} The most marked increase in any electrocardiographic change occurs in the T wave in lead I, which becomes inverted, alone or in combination with inversion in other leads, in 20 to 40 per cent of cases.^{10 14} Practically all other changes are also increased in frequency but to a lesser extent.¹⁰

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CHAPTER 26

THERAPY OF CARDIAC DECOMPENSATION

WILLIAM D. STROUD AND JOSEPH A. WAGNER

INTRODUCTION

THE chief factor responsible for myocardial failure in advanced age is coronary arteriosclerosis. The etiology of this condition is still obscure and remains amongst the foremost problems in need of research and investigation with a view to reducing the morbidity and prolonging the life of mankind. It is debatable to what extent, if any, arteriosclerosis might be considered a natural involutionary change (See Chapter 29). If such is not the case and if it should be found amenable to prevention, it would appear that the span of man's life would be considerably increased. However, such speculation is still in the realm of phantasy, and it remains for us to understand why this

The efficiency of the heart in old age remains unexpectedly adequate despite the normal physiologic senescent change—probably because of the lesser demands in the way of physical activity, basal metabolism, and the like. As mentioned above it is difficult to draw the line between what constitutes normal senescent change and pathology. It is well known that the

ventricular walls. The heart appears more conical. The usual amount of epicardial fat is diminished and the coronary vessels are elongated and tortuous. Normally there is a degeneration of elastic tissue and the deposition of finely divided calcareous material in the tunica media of these vessels. This is arteriosclerosis and constitutes a pathologic change. Strangely enough these changes are not present constantly. Why arteriosclerosis is in some individuals highly selective, affecting possibly only the cerebral, coronary, or peripheral arteries, is quite unknown.

Other conditions, of course, contribute to or primarily cause cardiac failure. To be mentioned are (1) other chronic prolonged debilitating disease, (2) anemias, (3) nutritional deficiencies, (4) chronic pulmonary disease producing pulmonary fibrosis, emphysema, etc. Chronic valvular disease, the result either of an antecedent rheumatic valvulitis with superimposed calcification of the valves or of calcification of the valves occurring primarily as a pathologic entity, is occasionally the cause of cardiac failure as a result of the myocardial strain so produced. This statement is subject to some criticism because it is often true that coronary arteriosclerosis is a common associated finding at necropsy.

CHRONIC VALVULAR DISEASE

Calcification of the aortic valve with or without stenosis or insufficiency has long been known as a clinical and pathologic entity. Secondary calcification of a previously affected valve has been in some cases distinguishable from that which occurs primarily in the absence of rheumatic valvulitis or subacute bacterial endocarditis. This distinction has been possible when a clear history of rheumatic fever in childhood is obtained. In this situation at necropsy the heart is often found enlarged and dilated as a result of the complete or button-hole type of aortic stenosis. The valves are often found curled and thickened along their free margins with calcification taking place along the free margin of the valve and extending centrifugally, possibly involving the entire valve. *This picture is in contrast to that observed in the individual who without previous rheumatic fever develops aortic stenosis or insufficiency in the late fifties or sixties.* At autopsy the aortic valve may show calcification about the aortic ring with heaped up exuberances of calcified atheroma in the sinuses of Valsalva. This calcification usually fails to involve the free margins of the valve but complete involvement does occur occasionally. Very often it is impossible to determine clinically or pathologically whether or not the valve has been the subject of a previous rheumatic affection, and the debate continues between those who contend that calcification never occurs in an unaffected valve and those who believe there is little evidence to support the contention that the valve has been the site of previous injury by disease.

The diagnosis of this condition is made easily when the following classical findings are present: (1) *pulsus tardus*—a pulse slow to rise and slow to fall, (2) low pulse pressure, (3) a systolic thrill over the aortic area associated with a loud harsh systolic murmur transmitted to the vessels of the neck, and (4) the absence or diminution in the intensity of the aortic second sound. Symptoms of cardiac failure such as dyspnea, orthopnea, and ankle edema may or may not coexist. It has often been said that the diagnosis of aortic stenosis could not be entertained in the absence of an aortic thrill. Autopsy records reveal the fallacy of this dictum. Possibly the only sign or evidence to suggest this diagnosis is a systolic murmur at the aortic area. The character of the pulse is dependent upon the pulse pressure. Therefore the character of the pulse may be considerably different if a degree of aortic incompetency with a wide pulse pressure is present. The thrill is probably dependent on the state of the myocardium, and if myocardial failure is present the thrill may be absent.

The electrocardiogram is of no help in the diagnosis of valvular conditions. Calcified valves are not infrequently noted on x-ray or fluoroscopic examination.

Calcification of the mitral valve occurs, especially of the anterior leaflet, either as an extension of the general tendency to calcification or secondary to a previous rheumatic valvulitis. In the first instance it may be responsible for the production of a systolic murmur but seems to be in no way the cause for unusual myocardial strain leading to cardiac failure.

MANIFESTATIONS OF CONGESTIVE FAILURE

Whatever the mechanism, i. e., whether it be increased (pressure theory) or impaired renal fu

• • • Failure

the kidney (the so-called forward failure theory), the fact remains that the symptoms of heart failure have long been identified as those of right or left ventricular failure. The most common sign of this condition is cardiac enlargement, the result either of cardiac dilatation or hypertrophy or both. The cause in older age groups is usually hypertension with arteriosclerosis of the coronary arteries, myocardial infarction, or simply coronary narrowing resulting in insufficient delivery of the required amount of blood to the myocardium.

Left ventricular failure produces dyspnea and particularly orthopnea. Paroxysmal nocturnal dyspnea is sometimes complained of either with or without pain. As such this symptom is not easily differentiated from angina pectoris. Passive congestion of the lungs ensues and is usually accompanied by cough, sputum, and even pulmonary edema.

Right ventricular failure, usually caused by either the passive pulmonary congestion induced by the failure of the left ventricle or by primary pulmonary disease such as chronic fibrosis and emphysema, manifests itself by engorged head and neck veins, enlargement of the liver, ascites, pretibial edema, and sometimes generalized anasarca.

TREATMENT

The principles of treatment of myocardial failure in the aged differ little from those attending the treatment of any disease in any individual no matter the age. Every good doctor must be understanding of the emotional reactions of his patients if he is to gain their confidence to the degree that they will be guided by his advice. The aged individual, infirm and insecure, realizing perhaps that his time is drawing short, is apt to be unnecessarily apprehensive concerning the gravity of his situation. He needs reassurance and guidance perhaps more than do the young who feel quite certain of their recovery. Even more understanding is required of the aged individual who, being ill and uncomfortable for long periods, becomes discouraged and morose. His depression grows deeper with each day of continued confinement. His faith in the medicines administered grows less and less. The doctor and the family begin to feel the hopelessness of the situation and the total effort becomes directed toward keeping the patient quiet until the end comes. This type of situation may be a very long drawn out affair and medicines may meet only a small part of the patient's needs. Those surrounding him must strive to make the patient feel their interest in his recovery. More than ever he requires attention and devotion. In small ways and within the physical and mental limitations imposed by the sickness he must be urged to do useful things. To allow him to be continuously alone, detached from family ties, useless and a burden, is to increase the difficulties attending his care.

Rest. It is not very often that we now think in terms of absolute bed rest for our aged patients. Absolute bed rest, cautiously used, may occasionally be required in severe congestive failure but the situation must be reviewed from day to day. This type of treatment appears to favor the incidence of phlebotrombosis with pulmonary emboli, pneumonia, etc. The wiser course appears to be something less than this, i.e., restricting the activity of the individual so that all unnecessary walking is eliminated. Permission is granted to have meals in a chair beside the bed and for the use of a commode rather than a bedpan. Good nursing care with much attention and assistance is

essential. Often it is wise to permit the patient his long ingrained habits such as early morning coffee, cigar and paper reading late at night and so on.

Sedatives. The proper and understanding use of these drugs contributes much to the satisfactory care of the patient in congestive failure. Nothing gives so much rest and opportunity for recovery as do narcotics to the orthopneic, dyspneic, cyanotic, restless and apprehensive patient. However, they must be used with great caution and prudence. We believe it may be better to give older people a lesser dose of narcotic than one might ordinarily prescribe and have it repeated later if necessary rather than to give more than can be tolerated on the first dose. Sometimes it takes surprisingly little to accomplish the desired results. Morphine sulfate $\frac{1}{8}$ to $\frac{1}{4}$ grain either with or without atropine sulfate $\frac{1}{15}$ to $\frac{1}{100}$ grain has been recommended. In recent years digitalis has been non-toxic, but its undesirable side

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efficacious and in

some quarters is said to be non-habit forming. Any drug which is chosen may have to be repeated frequently for a period of days or weeks and this knowledge helps to guide selection. Hypnotics such as nembutal $1\frac{1}{2}$ grains or seconal $1\frac{1}{2}$ grains at bedtime are often helpful to induce sleep. Phenobarbital $\frac{1}{4}$ to $\frac{1}{2}$ grain administered four times daily helps to allay some of the restlessness that attends illness and limitations. (See p. 34.)

Sodium salts of bromide, phenobarbital and the like are to be avoided in heart failure with water retention. It is not uncommon to find a patient receiving sodium bicarbonate in peppermint water administered regularly for flatulence while he is at the same time on a salt restricted diet because of edema.

Digitalis. In the administration of this most important drug there is still little to be added to what was said by Withering in 1785 *viz.*, "Let it be continued until it acts either on the kidneys, the stomach, the pulse or the bowels; let it be stopped on the first appearance of any one of these effects." This drug is most efficacious in cases of rapid auricular fibrillation or flutter wherein its action on the cardio-inhibitory mechanism aids in control of the ventricular rate by blocking the number of impulses transmitted to the ventricle from the auricle. In addition to this effect it also improves myocardial tone through direct action upon the myocardium and thereby improves the circulation. Rapid auricular fibrillation is not too common in aged subjects because of the degenerative changes such as fibrosis which interfere with auriculoventricular conduction. The more advanced the physiologic senescent changes the less beneficial are the effects of digitalis. In addition, the greater the degree of myocardial failure the greater the need for full therapeutic dosage of the drug. It is under such circumstances *viz.* where the margin of safety is narrow and where full therapeutic dose is essential that it is difficult to estimate the amount of the drug that is tolerable. The most common toxic effect noted is nausea and vomiting which may have been preceded by anorexia, dizziness, sweating, etc.

However, in the presence of severe myocardial degeneration the first evidence of toxicity may be arrhythmia. Premature contractions, auricular or ventricular in origin, appear. These premature contractions may arise from numerous foci. Their appearance suggests increasing myocardial irritability from the effects of digitalis. Because they may be the forerunner of

more serious forms of arrhythmia, such as paroxysmal auricular tachycardia or ventricular tachycardia, the patient must be observed very closely. Auricular fibrillation may be a toxic arrhythmia caused by digitalis. If the premature contractions were present before digitalis was administered they may disappear under its administration or they may become more numerous, seriously interfering with the establishment of compensation. Partial heart block may be increased to a complete block by digitalis. This development is attended by an unfavorable clinical response. On the other hand, very often the degree of heart block remains unchanged and clinical improvement follows the effects of digitalis upon the myocardium.

In the absence of digitalis in therapeutic dosage administered within the previous two weeks it is usual for a patient to require 0.9 to 1.1 gm. of whole leaf administered within twenty-four hours in divided dosage as a digitalizing dose. Thereafter the patient may be given 0.1 gm. twice daily until the effects of the drug are noted. More often it is unnecessary to digitalize a patient so rapidly, 0.1 gm. twice daily for a period of one week can be prescribed. By this method one has a greater opportunity to observe the patient for undesirable side effects. It is a slow method, but in introducing a very necessary drug to a patient who will in all likelihood require it for the rest of his days it is better to avoid unpleasant reactions which will give him a fixed aversion to carrying on with it. Once the patient is considered digitalized, a daily dose must be prescribed sufficient to maintain the effects already secured. This varies from individual to individual depending somewhat on the weight, but more often depending on the state of the myocardium. Usually 0.1 gm. administered from five to seven times weekly will suffice. Occasionally the patient will require as much as 0.2 gm. or as little as 0.033 gm. per day.

The tincture is little used because of difficulty in obtaining standard dosage (drops and minims are easily confused) and further because of instability of the product if allowed to stand about for long periods of time.

Much effort has been made in recent years to obtain a purified derivative of whole leaf digitalis which can be measured by weight rather than requiring the assay method to determine its potency. Digitoxin, now available in pure form, is such a product. It appears to produce effects similar to those of whole leaf digitalis, including the toxic reactions. For this reason the patient requiring it must be carefully observed in the same manner as though he were taking whole leaf digitalis. Perhaps there are a few patients in whom it is less apt to produce nausea. The average patient requires 1.2 mg. of digitoxin as a digitalizing dose when administered over a twenty-four-hour period. The daily maintenance dose is approximately 0.2 mg. daily. However, as with whole leaf digitalis, these figures are but the average requirements and *each patient must be individualized* and doses determined by the effects of the drug.

Rarely is it necessary to administer digitalis or digitalis-like drugs intravenously. All too often the circumstances under which this is done are considered emergent, but when the situation is reviewed subsequently it is found that proper indications for the use of this drug were lacking. Before digitalis is given intravenously one should be quite certain whether the patient has recently been taking digitalis or whether the patient has had a recent coronary occlusion. The drug is not administered by subcutaneous or intramuscular routes because of delayed absorption. Given by these routes it is irritating and the tissue reaction is considerable. When given intravenously it is usually

under the following circumstances (1) the patient is unable to take anything by mouth because of complicating gastrointestinal disease, (2) the patient is in severe cardiac failure with pulmonary edema, or (3) the patient is vomiting incident to congestion of the liver. Under these circumstances we have found the intravenous administration of a solution extracted from digitalis (digalen) effective. To digitalize we have usually given 8 cc (equivalent to 0.4 gm) initially, followed by a similar dose four hours later if it appears necessary and if the patient is still unable to take the drug by mouth. If gastrointestinal complications are present the daily dose of 2 cc or 0.1 gm may be given by this method.

We have not felt that any additional benefits were derived from strophanthin, ouabain, and the like. These drugs act quickly when administered by vein but it is doubtful that they exert any more rapid action or any more effective action than a solution of digitalis if the latter is given in a similar digitalizing dose.

Diuretics If the patient fails to respond from cardiac failure after proper rest, digitalization and salt restriction, a diuretic is in order. There are numerous drugs capable of exerting diuretic action, such as ammonium chloride, urea, xanthine products, and the mercurials but it is only the latter two groups and in fact only the mercurials, that are presently used when diuresis is urgent. The first two, ammonium chloride and urea have been given up largely because the quantities required to produce results cause gastrointestinal disturbances in many patients. Xanthine products are often prescribed before resorting to the more effective but possibly more dangerous mercurials. The additional effects of the xanthines, such as distinct coronary dilating properties, are desirable although if given in maximum dosage in order to produce such results they not infrequently cause cerebral excitation sufficient to disturb the rest of the patient. We commonly use purital (A. J. Parker Co., Philadelphia) 0.5 gm three or four times daily. Calpurate or theocalcin in the same dosage is also useful. Sometimes a better diuretic effect can be obtained by giving 1.0 gm three or four times daily on three successive days of each week.

If these measures fail and the patient continues to exhibit evidence of myocardial failure, such as dyspnea with rales present at the bases or ankle edema, it is usually wise to resort to the mercurials. It has been reported that death has occurred from mercupurin and similar compounds, when given intravenously. These fatal reactions are of unknown cause and have been said by some to be speed shock reactions from too rapid administration. Others suggest they are allergic reactions. Fortunately such reactions are rare for this is a most important group of drugs. It is advisable to be extremely cautious in applying the mercurial diuretics to patients with chronic nephritis whose urinary specific gravity is relatively fixed at 1.010.

For the reasons stated above we have recently elected to use mercurhydnn

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to the best of our knowledge. When given intramuscularly the diuresis is less spectacular in onset and possibly slightly less effective. (The devastating diuresis of intravenously administered mercurials is often dangerous. See p. 597.) The effects may persist for a longer period of time. It is usually given

in the morning so that the diuretic effect is largely dissipated by bed time. The injections are best given daily until no further weight reduction occurs, and then every other day or every third day and so on as frequently as is necessary to maintain the reduced weight. Sometimes it is sufficient to give the drug once every week or two. These drugs can be continued for an unlimited period of time and we have known those who received mercurials weekly or biweekly for over ten years.

Recently we have employed mercuzanthin tablets with favorable results in some patients who previously required weekly injections of mercupurin. These tablets can be given three times daily until diuresis occurs and then one tablet daily thereafter. If the necessity for diuresis is not so acute they may be given as one tablet daily and the diuretic effect remains unnoticed until several weeks have passed. As said before this seems to be a very satisfactory arrangement in some patients. In others the effect of the mercury on the gastrointestinal system mitigates their use.

Diet. The diet should be highly nutritious, easily assimilable, and low in cellulose, which so often is responsible for gas production. The latter causes discomfort and increases dyspnea by elevating the diaphragm. The most important factor in the diet in cardiac failure is to reduce the salt or more particularly the sodium intake. Both immediately prior to the development of edema and concurrent with the presence of cardiac failure the kidney is unable to handle the salt and water excretion as it normally does. Sodium ions are retained in the tissues and they in turn retain water which eventually becomes apparent as edema. It is therefore imperative to reduce the sodium intake below 2.0 gm daily. Most patients with impaired renal function are able to effectively handle this limited amount.

In severe cardiac failure this goal is easily attained by means of the Karell diet which is simply 7 ounces of skimmed milk four times daily. This diet eliminates any confusion concerning salt. After several days the diet must be increased to include jellies, junket, custards, etc., and later on meat, vegetables, and fruit, may be added, but it is wise to have patients avoid all salty foods such as salted butter, salt fish, bacon, ham, and the like. To date we have not learned of a completely satisfactory salt substitute. There are many so-called substitutes available, but for the most part they simply substitute other acid radicles for the chloride and permit the sodium to go unaltered.

Fluids were formerly reduced in failure to 1 liter daily. Presently we feel the patient may have fluids *ad lib* provided the salt content in the diet is reduced, as mentioned above. We have not flooded our patients as advised by some. It is not uncommon for the edematous person to avoid fluids, feeling that they were the cause of his trouble. He must understand that approximately 2 liters daily is necessary in order to maintain adequate renal elimination. Otherwise retention of urea and acid metabolites occurs which complicates the problem.

Other Measures. Oxygen is exceedingly useful in the patient with cyanosis and dyspnea and also in the patient with continuous substernal distress following a coronary thrombosis. It can be administered as either a 50 to 60 per cent concentration in an oxygen tent or by nasal catheter. This form of treatment can be continued for days or weeks without untoward effects.

Venesection is no longer carried out as extensively as formerly, but

occasionally it still proves helpful in patients with acute left ventricular failure leading to pulmonary edema. These patients most often have hypertensive, arteriosclerotic cardiovascular disease. Evidence of right ventricular failure may or may not be present. Removal of 250 to 500 cc of blood from the arm vein is often followed by rapid improvement. In patients with obstinate failure and edema, unimproved after the above measures have been thoroughly tried, the *removal of fluid* from serous cavities when such is present often produces symptomatic relief. Southey tubes, used so frequently in the days before mercurial diuretics, still have a place in treatment of the patient who has not responded or who for reason of idiosyncrasies is unable to take the more modern drugs. Occasionally the removal of large quantities of edema fluid in this manner is enough to tip the scales in favor of the patient and compensation begins to return.

Times change and with the times our attitude toward cardiac failure changes. Prolonged rest and marked restriction of fluids are no longer so necessary. Better standardized digitalis preparations are becoming available. More than ever the physician has the opportunity to demonstrate his effectiveness in the management of the sick.

CHAPTER 27

ANGINA PECTORIS, MYOCARDIAL INFARCTION, AND ACUTE CORONARY FAILURE

WALTER S. PRIEST

INTRODUCTION

It is doubtful if "normal" age changes, as described in Chapter 3, could cause impairment of cardiac function other than decrease in reserve power with consequent reduction in the scope of sustained physical effort. On the other hand, coronary artery disease with all of its sequelae is probably the most common clinical entity encountered in the geriatric period. This certainly becomes true if we include the local and secondary effects of hypertensive disease. While the etiology of atheromatous change in the arteries is as yet not clearly understood, the current tendency is to consider it a disease process, rather than a part of "normal" aging. This more satisfactorily explains the common occurrence of coronary arterial changes out of proportion to the apparent or chronologic age of the individual.

While the diagnostic problems resulting from coronary vascular lesions are the same regardless of the attained age, prognosis and the problems of management vary somewhat depending upon whether the patient is in middle life or has reached that period commonly called "aged."

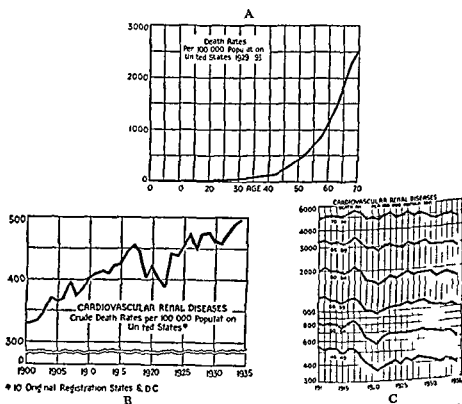
INCIDENCE

The death rate from cardiovascular renal diseases (243.8 per 100,000 in 1945) continues to exceed all others. However, the decline which began in 1934 has continued for all age groups, and ranges from 1.7 to 8 per cent depending on the particular age group studied. (These data were generously furnished by the Metropolitan Life Insurance Company, New York.) It is encouraging that the percentage of decline increases steadily with each succeeding decade from age thirty-five to seventy-five. From the evidence at hand it is impossible to say whether we are making some headway in the prevention of atheromatous disease. Perhaps we are more skillful in prolonging the life of patients so afflicted until death ensues from some other cause. Statistics show increasing numbers of persons alive and economically useful ten and more years following acute myocardial infarction. This continued improvement in the death rate from cardiovascular renal diseases should stimulate increased research. In contrast to the declining death rate from cardiovascular renal diseases as a whole, the certified death rate from coronary artery disease has increased from 65.6 to 72.9 per 100,000 during the past two years. Most observers agree that this is due to changes in nomenclature on death certificates and to the lessened use of such diagnoses as "myocarditis" and "chronic nephritis."

In spite of the declining death rate, the increasing number of persons over age forty will probably mean an increase in the actual number of cases of cardiovascular renal disease for some time to come.

TERMINOLOGY

Until about thirty five years ago almost any chest pain in the precordial region was called angina pectoris particularly if the pain radiated upward to the left shoulder and down the left arm. Pain of great severity and long duration accompanied by shock and repeated attacks of pain even while the patient was at rest were called status anginosus. If the patient died in such an attack his death was attributed to angina pectoris. Dock¹ in 1896 observed and described acute myocardial infarction at autopsy. The observations of Herrick² in 1912 set forth the clinical picture of acute myocardial infarction.



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 1 - a beginning with
 2 - rate in
 3 - of the

and paved the way for its recognition apart from the angina of effort. Attention was focused on sudden occlusion of the coronary arteries and a voluminous literature dealing with all phases of this problem has accumulated. It is a common feature of the fact that the common feature are all regulation and consequent

middle life or past is attributed to a basis of coronary occlusion appears on the death certificate in spite of

the fact that sudden death may be caused by other things, for example, pulmonary embolism

"He had a coronary" is the offhand diagnosis repeatedly heard in reference to a patient having an attack of chest pain of any great severity or duration. Physicians well versed in heart disease are guilty of such loose terminology although their diagnosis of the condition may have been most accurate. Confusion has been occasioned by attributing synonymous meaning to the terms "coronary thrombosis," "coronary occlusion," and "myocardial infarction." From the results of postmortem studies,³ the writer has been convinced for years that there is no recognizable clinical syndrome of coronary thrombosis or coronary occlusion as such. The excellent studies of Blumgart and Schlesinger⁴ have confirmed this impression. Inasmuch as coronary thrombosis and occlusion can occur without myocardial infarction and infarction can occur without coronary thrombosis or occlusion, the terms "thrombosis" and "occlusion," whether "acute" or not, should be left for the use of pathologists.

Not infrequently, a patient may have a series of attacks of pain, with or without evident precipitating cause, lasting an hour or more each time. These attacks may have some of the features associated with myocardial infarction. Any one or all of them will be referred to as "another coronary" by the uncritical observer, yet at autopsy only one infarct is found. At times only severe coronary lesions with atheromatous occlusions or a single recent thrombus may be found. Gross infarction may or may not be present. Attempts to find an appropriate clinical label for the attacks not associated with myocardial infarction have led to the use of other terms, such as coronary insufficiency, coronary failure, coronary accident, and "heart attack." While it may be that some noncommittal term such as "heart attack" may have to be used in talking to relatives until careful study permits as accurate a diagnosis as possible, such an indefinite term or the equally vague one of "coronary" should never be used as the final diagnosis, nor should it be used in professional discussion. Likewise it is inaccurate and unnecessary to attribute synonymous meaning to infarction and coronary occlusion.

The most recent studies suggest *three clinical pictures* resulting from inadequate coronary circulation to which may be given terms having reasonably accurate physiologic and pathologic meaning. While some minor difference of opinion as to details still exists, an understanding of the essential features of each clinical entity and the meticulous use of the appropriate term will greatly improve accuracy of diagnosis, make for better visualization of the probable anatomic lesions, and do away with confusion in terminology. This grouping is as follows:

1 *Angina Pectoris* This comprises transitory attacks of pain produced by some form of effort or effort equivalent and promptly relieved by rest or nitrites.

2 *Acute Myocardial Infarction* There is sudden onset of pain, usually severe but not necessarily related to a recognizable precipitating cause, accompanied by shock and fall in blood pressure and followed by fever, leukocytosis, increased sedimentation rate, and characteristic electrocardiographic changes requiring days or weeks for recovery.

3 *Acute Coronary Failure* In between the extremes of (1) and (2) is the type of attack which has some of the features of both but is without the exact

picture of either. The concept of acute relative insufficiency of the coronary circulation of longer duration than obtains in simple angina pectoris, but which passes off before massive infarction occurs, best seems to fit this syndrome. The term "acute coronary failure" indicates a better understanding of what actually takes place and prevents the misapplication of the terms "coronary thrombosis" or "coronary occlusion."

ETIOLOGY

The basic etiology of angina pectoris, myocardial infarction, and acute coronary failure is the same as the etiology of the vascular changes in atherosclerosis and hypertension. Precipitating factors may be recognized in the various clinical episodes, but these do not constitute the etiology of the underlying disease without which the various clinical attacks would not occur. As yet it is not known what causes atherosclerosis or arteriosclerosis (see p 466), nor is it known why these lesions may occur in the third decade or even earlier in some people, while others live well past eighty without significant intimal or medial changes. Heredity, stress and strain of modern life, diet, toxins, infections, endocrine disturbances, obesity, allergy, disturbances in cholesterol metabolism are among the factors thought to be of etiologic significance. Yet no single factor or combination of factors seems to apply to all cases. In the light of present knowledge it seems that inheritance plus something in the individual's life determines whether he will develop vascular lesions early or late, or to any significant extent at all.

The average age at which clinical symptoms of coronary vascular disease first make their appearance is about fifty-five, but symptoms may appear from the second decade on. Men are more frequently victims than women. First symptoms are more apt to appear during the winter months. Unskilled urban workers and professional people show the highest incidence while agricultural dwellers and workers have the lowest, by a wide difference. Other occupations and strata of life fall in between these two extremes.

Since so little is known of the etiology of this most important disease little can be accomplished in the way of prevention. It is hoped that the modern interest in geriatrics is a step toward the ultimate solution of the problem.

PATHOLOGIC ANATOMY

The anatomic common denominator in angina pectoris, coronary failure, and myocardial infarction is a damaged myocardium resulting from varying degrees of interference with the coronary blood flow. In some cases of angina pectoris it may be that the damage results entirely from long continued overstrain of the myocardium although personally no examples of this have been observed at the postmortem table.

Interference with Coronary Circulation This may be caused by atheromatous narrowing of the arteries (intimal sclerosis) arteriosclerosis (medial hypertrophy) with narrowing of the lumina, or both conditions may exist in the same heart. Thrombi may develop at the site of atheromatous plaques or ulcerations. These may rapidly occlude the vessel completely or remain for a time as mural thrombi, narrowing, but not completely occluding the lumen. Eventually, fresh thrombus formation takes place with complete occlusion. Once formed, the thrombus may increase proximally and/or distally, progressively shutting off branches of the occluded vessel.

Intimal hemorrhage into an atheromatous plaque may protrude the area into the lumen to the point of complete occlusion or induce thrombus formation at the site. Finally, a vessel may be completely occluded by the slowly increasing thickness and size of the atheromatous plaque without thrombosis taking place.

Myocardial Lesions These consist of fibrosis, especially in the perivascular spaces. This connective tissue is usually poor in nuclei and many of the fibers are hyalinized. Fragmentation of the myocardial cells may occur. These changes vary considerably in extent, are sometimes visible to the unaided eye as white streaks in the cut surface, and at other times require microscopic sections for their demonstration. The result of arteriolar sclerosis alone is diffuse fibrosis and myocardial fragmentation of varying degree analogous to the lesions seen in nephrosclerosis (see p. 74). Obliterating endarteritis has not been observed.

When the vascular lesions result in *myocardial infarction*, various gross and microscopic pictures are seen, depending on how long the patient survives after the infarction. Both old and recent infarcts may be present in the same heart. In very recently infarcted areas a *reactive process* consisting of hyperemia, capillary enlargement, extravasation of red blood cells and invasion of polymorphonuclear leukocytes may be present. It is of clinical importance that complete healing (fibrous replacement) of an infarct may not take place for many weeks. Old infarcts are represented by *fibrous tissue scars*. These may occur as thin areas of the ventricular wall with resultant aneurysmal bulging. Occasionally recent, organizing, and healed infarcts are seen in a single area. This represents a series of insults or, more likely the progressive deprivation of the area caused by slowly developing atherosclerotic plaques or the extension of a thrombus. Very little attempt at regeneration of the myocardial fibers is seen in the vicinity of infarcted areas, but apparent hypertrophy may be observed. Mural thrombi may occur at the site of infarction, giving rise to the hazard of embolic phenomena in the peripheral or pulmonary circulation. Whether the anterior and apical portion of the left ventricle is more frequently the site of infarction than the posterior and basal portion is a matter of some controversy. The preponderance of carefully compiled evidence seems to indicate no great difference. The right ventricle and the auricles are almost never the site of myocardial infarction.

Collateral Circulation Of vital clinical importance is the development of anastomotic or collateral circulation in the coronary arterial system. This takes place as life advances, apparently in preparation for the almost inevitable impairment of age. It seems that the more severe the vascular lesions the more adequate is the collateral circulation, as if Nature were doing everything possible to minimize the results of severe narrowing or complete occlusion. In fact the work of Blumgart and Schlesinger⁵ indicates that the anastomosing channels do not exceed 40 micra in diameter regardless of the patient's age unless the coronary arteries are narrowed or there is some unusual work demand on the heart. That age alone is not the factor in the development of significant collateral vessels is indicated by their presence as early as the third decade in a heart which is the seat of severe coronary sclerosis. Other possible sources of blood supply have been described, particularly the thebesian vessels and some extracardiac anastomoses. Whatever the source, it cannot be doubted that a given area of myocardium in the aging

or aged period has more than one possible blood supply. In spite of this, one is frequently amazed at the extent and severity of vascular and myocardial lesions with which a heart has continued to function for considerable periods of time.

The *protective value* of these collateral channels is indicated by the fact that, in order for gross myocardial infarction to occur, severe interference with the blood flow in at least two main stems or major branches of main stems must be present.^{3,4} Thus coronary occlusion may occur without infarction, if the flow in the other stems is not impeded by significant atherosclerotic narrowing or previous occlusions. The infarcted area is not necessarily in the region receiving its principal blood supply from the vessel most recently occluded. Figure 74 illustrates this point. The branch of the left coronary artery normally supplying the infarcted area had previously been occluded by calcified plaques. Yet infarction did not occur until thrombotic occlusion of the right main coronary stem deprived the area of the blood supply it had been receiving from this latter vessel. An important clinical corollary of this

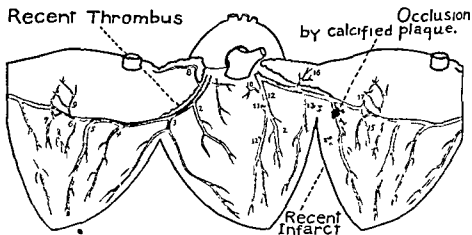


Fig 74 Pathology consequent to coronary arterial disease

anatomic fact is that when a diagnosis of myocardial infarction is justified, serious interference with the blood flow in two coronary branches must be visualized.

Except for the premise outlined above no accurate idea of the extent of the vascular and myocardial damage can be obtained from the clinical findings. One patient may have a most extensive atheromatous involvement of all coronary branches plus generalized myocardial fragmentation and fibrosis, yet have few clinical signs of myocardial insufficiency or structural damage. Another patient may have only localized atheromatous areas in the larger branches and relatively little myocardial fibrosis with equally few clinical signs, yet succumb rapidly from occlusions, or infarctions. Without hyper-
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present unless signs of infarction develop. The patient with angina pectoris who does well on proper management, and the patient who recovers from

acute myocardial infarction without significant residual symptoms, may be assumed to have less extensive vascular and myocardial damage than those who do not do so well

CARDIAC PAIN

Pain is the common symptom of the three clinical pictures resulting from impaired coronary circulation and the typical location and radiation of the pain is substernal or precordial, extending to the left shoulder and down the left arm. Many atypical locations and radiations are encountered.

Location of Pain. The following table⁶ gives the location and radiation of pain in 52 consecutive patients on whom a diagnosis of angina pectoris, acute myocardial infarction, or acute coronary failure was justified

I Substernal—23

- 1 Without radiation—7
- 2 To right and left arm—7
- 3 To left chest and left arm—2
- 4 To back—2
- 5 To epigastrium—1
- 6 To midline posteriorly to neck, to r and l shoulder—1
- 7 To throat—1
- 8 To right and left arm and jaw—1
- 9 To left side of neck, shoulder, arm and l scapula—1

II Precordial—9

- 1 Without radiation—2
- 2 To left shoulder and arm—4
- 3 To entire chest—2
- 4 To interscapular region—1

III Epigastrium—9

- 1 Without radiation—1
- 2 To right shoulder and arm—3
- 3 To left arm—3
- 4 To right and left arm and around chest—1
- 5 To right upper quadrant of abdomen—1

IV Miscellaneous—11

- 1 Right chest to right shoulder and arm—3
- 2 Both sides of chest anteriorly to r and l arm—1
- 3 Left shoulder—1
- 4 Left shoulder to upper chest—1
- 5 Left elbow to shoulder—1
- 6 Right upper quadrant of abdomen only—1
- 7 Jaw to chest—1
- 8 Left little finger up arm to precordium—1
- 9 Both wrists (band like) jumping to right and left chest anteriorly without involving arms—1

Origin of Pain That chest pain may result from other causes than a damaged heart is obvious. Yet when almost any chest pain appears in an individual past middle life it seems common practice to diagnose angina pectoris or coronary disease without further ado. Since this may be unfair to the patient and his future such diagnoses should be made only after investigating other causes for chest pain and ruling them out. This is particularly true of

pain of extracardiac origin manifested through the same skin segments as that of cardiac pain

In arriving at the true cause of chest pain, the following outline, modified from Gunther,⁷ will be helpful

I Nonorganic Pains

1 Functional and Psychic Pattern Pains

- The 'red hot' needle, nipple point pains (Kilgore)
- The needle point pains at the breast in vasovagal attacks (Albutt)
- Precordial aching of neurocirculatory asthenia
- Occupational back pains of nurses typists etc
- Circular pains occupying part of the arc of the circle at the base of the breast or around the nipple usually under, and lateral to the breast

II Organic Pains Involving the Viscerosensory Reflex

1 From the Mediastinum

- Angina pectoris (angina of effort)
- Acute coronary failure
- Myocardial infarction
- Obstructive lesions of the esophagus
- Cardiospasm
- Diaphragmatic hernia into the mediastinum
- Acute pericarditis and acute mediastinitis

2 The Pleural Reflex

- Pleurisy in acute pulmonary and pleural inflammations
- Pulmonary embolism and infarction
- Spontaneous pneumothorax
- Shoulder and chest pain in diaphragmatic pleurisy as in acute inflammations and tumors (including subdiaphragmatic lesions) The left hypochondrium pain associated with aerophagia and distention of the colon

III Root Pains (the radicular syndrome radiculitis segmental pain) or Organic Pains not Involving the Viscerosensory Reflex

- Arthritis of the dorsal and cervical spine
- Cervical rib
- Meningeal and nerve root tumors
- Tuberculosis of the vertebrae
- Erosions of the vertebrae by aneurysms lymphoblastoma and other new growths
- Destructive lesions in the intervertebral discs and vertebrae
- Compression fractures of the vertebrae (Kummell's fracture)
- Acute infections of the nerve roots and syphilis of the nerve root without bony changes (Dejerine)
- Acute virus infections as in herpes zoster
- Tabes dorsalis
- Scoliosis and muscle fatigue with static and postural root pains without bone changes

It will be seen that in many instances the differentiation is not difficult if one will only bear in mind the possibilities. Pains which offer more difficulty in differentiation from cardiac pain are those grouped under III of the outline, particularly the root pains and the gastric, mediastinal, pericardial and pleural reflex pains in II

As an example of the problem presented by chest pain involving the viscerosensory reflex, the following case may be cited

A woman of sixty three was awakened about midnight by severe substernal pain associated with nausea, vomiting, cold sweat and pallor. The pulse was rapid but of good quality. The heart tones were regular and of good quality. The blood pressure reading was of no help since previous pressure levels were unknown. Morphine was required for relief. There was a history of previous attacks of epigastric distress thought to be caused by gallbladder disease. Electrocardiogram later in the morning showed no definite diagnostic deformities. The leukocyte count was not elevated. A second electrocardiogram the following day showed no change. Temperature was not elevated. While these findings pointed away from acute myocardial infarction, opinion as to the possibility of acute coronary failure was withheld. Recurring chest pain with nausea continued. X ray revealed a gastric diaphragmatic hernia which reduced spontaneously followed by cessation of all symptoms. To have misdiagnosed this as angina pectoris or a coronary accident would have been a grave injustice to the patient as she is an active self supporting woman whose current mode of life need not be restricted.

Other causes of pain involving the viscerosensory reflex, aside from coronary disease and myocardial damage can almost always be determined by careful physical and laboratory study.

Because of the frequency of spinal arthritis in the age groups where cardiac pain is most common, the physician is very often called upon to differentiate between *radicular pain* and *cardiac pain*. The differentiation is not difficult if a detailed history is taken and a careful examination made without prejudice because of the patient's age. *Cardiac pain* is usually felt beneath the sternum. Whether the sensation is only a mild uneasiness or whether it is severe enough to make the patient stop all activity for fear the pain will become worse, it is usually described as being within the chest. Asked to locate his distress, the patient is usually vague and places his hand in the general region of the sternum or precordium. *Radicular pain* is most apt to be described as being on the surface of the chest and the patient points to or draws his finger over a fairly definite area. It does not have a constant relationship to effort. It is true that movements of the spine may bring it on, hence it may be related to walking or some other form of exercise. Careful questioning, however, will reveal that it also occurs at rest or sitting or lying in certain positions. If the pain radiates, valuable additional information may be obtained. The radiation of cardiac pain does not follow the zone in which it starts but jumps to neighboring zones. Thus pain felt in the sternal region (D2-D7) commonly radiates to the forearm and fingers (D1-C8) or to the shoulder (C4-C5). In so doing it does not complete the pathway to the spine of the peripheral segment in which it originated. Radicular pain, while it may arise in any or all of the above mentioned areas, radiates in a band like manner from front to back or vice versa and does not skip from one peripheral zone to another without completing at least part of the distribution of the original segment or segments. An interesting combination of both cardiac and radicular pain in the same individual illustrates these points.

A man aged seventy-six complained of indigestion for which he had taken various remedies without relief. He also had pain in the region of the left nipple and shoulder and down the left arm.

The "indigestion" pain was a vague uneasiness or pressure beginning in the epigastrium and extending upward beneath the sternum to the fourth or fifth rib where it spread to both sides but did not extend beyond the anterior axillary lines. (Note that it did not spread laterally at the point of origin.) It came on only with exercise (walking rapidly especially against a wind or in cold weather) and usually exercise after eating. It increased in intensity until physical activity was stopped.

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- Arthritis of the dorsal and cervical spine
- Cervical rib
- Meningeal and nerve root tumors
- Tuberculosis of the vertebrae
- Erosions of the vertebrae by aneurysms, lymphoblastoma and other new growths

without

Acute virus infections, as in herpes zoster

Tabes dorsalis

Scoliosis and muscle fatigue with static and postural root pains, without bone changes

It will be seen that in many instances the differentiation is not difficult if one will only bear in mind the possibilities. Pains which offer more difficulty in differentiation from cardiac pain are those grouped under III of the outline, particularly the root pains, and the gastric, mediastinal, pericardial, and pleural reflex pains in II.

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error. Since the prognosis and subsequent management of the two conditions is so different such an error does great injustice to the patient. Again the differentiation rests on careful examination, x-ray, and electrocardiographic studies. The electrocardiographic abnormalities produced by pericarditis are fairly typical and do not correspond to the patterns usually produced by myocardial infarction.⁸

Whether smoking of itself can produce cardiac pain is open to question. I have seen a few comparatively young men who complained of substernal or precordial ache, not precipitated by effort, following overindulgence in tobacco. No evidence of heart disease or other cause of the chest pain could be found and pain was not experienced under any other conditions. The attacks disappeared on stopping the use of tobacco. The tolerance to the effects of smoking varies greatly among different individuals but the use of tobacco is to be considered as a possible contributing factor to increased frequency or severity of attacks of cardiac pain in the elderly.

Seeing the patient in an attack or under circumstances which should produce an attack is helpful. A woman complained of attacks of chest pain thought to be cardiac in origin. However, her ability to climb twelve flights of stairs without inducing an attack definitely ruled out organic heart disease as the cause. The milder forms of cardiac pain may have no associated symptoms. If pallor, sweating, breathlessness, or nausea is present, however, the presumption is that the pain is cardiac or involves the viscerosensory reflex. Regardless of the intensity of radicular or nonorganic pain, these associated symptoms occur so rarely as to be negligible. Fear of impending doom (*angor animi*) is indicative of cardiac pain but it has not occurred in the writer's experience with anything like the frequency which older texts indicate. When cardiac pain is atypical in location and radiation it is more difficult to arrive at the correct diagnosis. If careful search for a local cause reveals nothing it is reasonable to assume that the pain is cardiac in origin. Admittedly there will be the occasional case where one is in doubt even after the most careful analysis.

A man of forty-two on whom a diagnosis of angina pectoris had been made came under observation six years ago. The onset, location, radiation, and other features of the attacks certainly seemed to confirm the diagnosis of the referring physician. Physical examination and laboratory studies failed to indicate an extracardiac cause for the attacks. Electrocardiograms were normal. Blood pressure fell after he ran up one flight of stairs. This seemed to confirm the possibility of some myocardial insufficiency. The possibility of a nutritional impairment was considered and high vitamin and mineral intake insured. After several months of this some decrease in frequency of the attacks resulted, but they did not cease altogether. While not overweight, he was a hearty eater of rich food. Of his own accord he decided drastically to reduce his carbohydrate and fat intake and eliminate highly seasoned food. This was followed by disappearance of the attacks and there has been no recurrence. Coincidentally, his business situation improved and he notes that for the past year he has been less tense and 'nervous'. The blood pressure response to effort is now normal. Here we have the features of pain apparently of the viscerosensory type and seemingly cardiac in origin ceasing, instead of gradually increasing, with no change in the electrocardiogram or other findings over a six year period. It is difficult to assume coronary sclerosis or structural myocardial damage as the basis. Some metabolic or functional disorder of the myocardium might be the answer.

It is impossible to cover in detail here all the diagnostic points to be borne in mind when confronted with the problem of chest pain. It is hoped

that the foregoing discussion will serve to stimulate more careful clinical and laboratory study of each patient in order that erroneous diagnoses of coronary disease with subsequent curtailment of activity and mental anxiety may be avoided

ANGINA PECTORIS

Clinically the term "angina pectoris" should be reserved for cardiac pain brought on by physical effort or emotional excitement, which subsides promptly on rest or the administration of nitroglycerin. The correct physiologic concept appears to be that of relative myocardial ischemia or anoxia resulting from a temporarily inadequate coronary blood flow. Various factors may produce this temporary inadequacy. A healthy young athlete may experience severe precordial distress at the end of a race, the maximum possible flow through normal vessels was inadequate for that amount of effort on the part of the myocardium. In the presence of any lesion or situation which reduces coronary blood flow, the myocardial effort for which the flow is adequate may be less than normal. The mere presence of the lesions, however, is not enough to produce attacks of angina pectoris. Severe degrees of coronary vascular lesions including complete occlusions are found post mortem in patients giving no history of angina pectoris. Some other factor is involved. This may be vascular spasm (vasomotor instability), toxins (such as tobacco), adrenalin effect (emotion), splanchnic dilatation (full meal) lowered blood pressure, low individual pain threshold, nutritional (vitamin) deficiencies, reduced oxygen concentration in the blood (anemia, high altitudes), accumulation of fatigue metabolites in the myocardium, inadequate collateral coronary circulation, and finally the extent of structural myocardial damage (fibrous replacement, scarring, fragmentation, and infarction). As Saphir³ has pointed out, it is conceivable that a damaged myocardium may have temporary periods of insufficiency—inability to meet the work load of the moment. The precipitating cause of the insufficiency may appear insignificant. Certainly the work load necessary to produce the insufficiency in a given heart varies. As a result of the myocardial insufficiency the cardiac output decreases and the blood supply to the myocardium is impaired. The effects of this momentary decrease of coronary blood supply may be responsible for the attack of angina pectoris. It has not been demonstrated that a clinical attack of angina pectoris (angina of effort) always means that a minute myocardial infarct has occurred. While it may be that angina pectoris will occasionally occur in an apparently undamaged heart, all hearts of patients middle aged or past with a history of angina pectoris, which we have seen at autopsy, have shown vascular and myocardial lesions. Practically, therefore, a diagnosis of angina pectoris in this age group is tantamount to

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extent of such damage and the frequency or severity of the attacks *vice versa*. Symptoms, physical signs, and the information gained from laboratory tests are not altogether reliable indicators of the extent of the anatomic changes. When a diagnosis of angina pectoris is made on a patient in the age group under discussion, all that can be said is that some degree of myocardial damage exists. Likewise some degree of coronary vascular disease

may be suspected. No idea of the prognosis in a given case can be had until there has been sufficient time and opportunity to study the patient's personality; his environment, both business and domestic, his pain threshold, the state of his general health and nutrition, and his response to simple rest. Sir James Mackenzie repeatedly stressed the clinical concept that an attack of angina pectoris means, first and foremost, an exhausted heart, but the fact of the attack tells nothing about life expectancy. When all factors contributing to the exhaustion of the myocardium are removed or corrected to the best extent possible, it frequently will be found that the anginal attacks disappear or become infrequent, although the structural myocardial damage or coronary lesions remain unchanged. This permits of a relatively optimistic prognosis as far as many years of useful life are concerned. On the other hand, if the attacks continue and even increase in frequency in spite of adequate management and the best possible correction of contributing factors, the prognosis is usually poor. Because of low pain threshold and neuro-emotional instability a patient may have a poor "work prognosis" yet live many years on a "retirement" basis.

The *electrocardiogram* is of relatively little value in prognostication. Entirely too much importance is attached to graphic records. Personal experience and the reported experiences of others indicate the limited value of graphic records in determining the prognosis of angina pectoris uncomplicated by recent or old myocardial infarction or predominant ventricular strain. Normal or nearly normal records are frequently obtained on patients having anginal attacks who live only a short time after the first consultation (see Fig. 75, b). Conversely, it is not uncommon to see patients whose electrocardiograms suggest considerable myocardial damage, yet who respond well to management and live for many years (see Fig. 75, f).

Finally, it must always be borne in mind that there is no way of accurately estimating the degree or extent of coronary sclerosis, if any, in a given case of angina pectoris. No matter how mild or infrequent the attacks or how favorably they may be affected by adequate management, the patient may succumb within a few months as the result of coronary occlusions or myocardial infarction (see Fig. 75, b). This possibility, however, does not justify pessimism in the physician's attitude toward the patient who responds well. In some instances, circumstances may make it advisable to discuss the possibility of sudden death with a business associate or a near relative. Rarely, if ever, is it desirable to discuss it with the wife or husband of the patient. Even with patients who do not respond well, optimism as to the outlook should dominate the physician's attitude. With a little common sense diplomacy the physician can bring the patient to keep his business and personal affairs in order and to refrain from those actions, such as driving a car, which might injure others in case of sudden death.

Diagnosis and Management. Obviously the first step in diagnosis is to determine whether or not the attacks of pain or pain equivalent are cardiac in origin. The differentiation of chest pain has already been discussed and it was noted that it is not always possible to determine the cause of the pain at the first consultation. Sometimes the pain is so obviously not cardiac that the patient may be reassured and permitted to follow his usual mode of life while necessary studies are being made. Where such is not the case, and since attacks of angina pectoris should be taken as an indication of myocardial

exhaustion or insufficiency, a patient should be at bed rest during diagnostic studies, although ultimately the heart may be found not to be at fault

Whenever possible the study should be carried out in a hospital, thus giving the physician opportunity to observe the effect of simple rest away from the patient's usual environment. Studies of renal function, metabolic rate, daily blood pressure and pulse levels, blood chemistry, together with fluoroscopy of the chest and other indicated x-rays, blood counts and serologic tests will give valuable information. Search for foci of infection should be made. Serial electrocardiograms will aid in ruling out acute myocardial infarction and acute coronary failure. As an aid in the diagnosis of angina pectoris *per se* electrocardiograms are of little value. Several observers have reported transitory changes during the actual attack. These changes are similar to those associated with acute coronary failure in that they consist most commonly of depression of the RST segment in leads I and II with changes in the amplitude of T. In view of the more recent terminology it may be that attacks accompanied by such electrocardiographic changes should be called acute coronary failure. In any event, as Barnes⁹ points out, "Positive electrocardiographic findings are to be anticipated in only about 50 per cent of patients who have an attack of angina pectoris and, if abnormalities are observed, their interpretation may be difficult and their significance in a given case may be open to question." In other words, the presence of changes during an attack, if properly interpreted, may aid in differential diagnosis, but the absence of changes means nothing. Also, as pointed out by Barnes, attempts to produce an attack by exercise or other means in order to record possible electrocardiographic changes are not without danger and are unjustifiable. After some thirty years' experience with the electrocardiograph, the profession is again becoming aware of the truth of Mackenzie's statement (at least as far as angina pectoris is concerned) that instruments of precision have but a limited sphere of usefulness in the examination of patients. It would be better not to use graphic records at all unless one is prepared to interpret them in correlation with the patient's history, physical findings, and the work ability of his heart (see Fig 75, d and g).

The effect of decreased oxygen concentration (relative anoxia) on the electrocardiogram has come into vogue recently as a means of differentiating "true" from "false" angina pectoris. The test is simple. A control electrocardiogram, using one or more precordial leads in addition to the standard limb leads, is made. The patient then breathes, through a Boothby mask, a mixture of 85 per cent oxygen and 15 per cent nitrogen for ten minutes. At the end of this time another electrocardiogram is made. If the two records show no change, the chest pain complained of is probably not cardiac in origin, or at least it is unlikely that significant coronary disease exists. If definite changes in RST and T, as noted above, occur it is strong presumptive evidence of coronary disease. The writer has used this test on several hundred subjects without serious effect. It has also been useful in estimating the exact significance of observed electrocardiographic abnormalities such as incomplete and complete bundle branch block.

If the diagnosis proves to be angina pectoris, the easy access to the patient during a hospital stay enables the physician to become acquainted with him through daily friendly conversation. Successful management of the patient with angina pectoris requires an intimate knowledge of his family

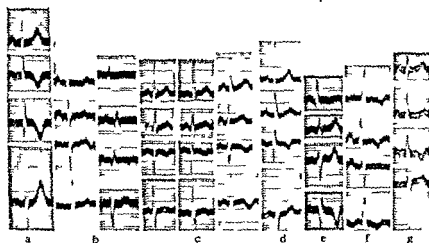


Fig 75 Electrocardiographic records (a) Q_T-T_1 type of record usually associated with posterior wall infarction. Cone shape inversion of T in leads II and III. Upright T in leads I and IV. (b) Male aged forty nine. First record made six months before infarction. Moderate hypertension, mild angina of effort. Note relatively slight abnormalities, chiefly those associated with left ventricular strain. Autopsy showed severe and extensive coronary sclerosis with marked narrowing of lumina which must have been present at the time this record was made. Second record, one of a series during a period comprising two separate episodes clinically indicative of infarction. Note striking difference in general appearance compared with the former record, but absence of significant Q and grossly inverted T. All records of the series were similar to this with only slight progressive changes. The small QRS persisted until death from fresh thrombus and infarction six months later. Autopsy showed large old infarcts anterior and posterior and a fresh infarct posterior. (c) Male aged sixty four. Nine days after mastectomy, sudden precordial pain, fall in blood pressure, fever, leukocytosis. Note progressive changes in T_1 and T_2 . This combination spoke against myocardial infarction. Autopsy showed pulmonary embolism with pulmonary infarct secondary to static venous thrombosis. No coronary sclerosis, no myocardial infarct. (d) Male aged seventy. Q_T-T_1 type of record suggestive of old myocardial infarct. Moderate hypertension. No angina. Able to walk three miles without discomfort. Similar record seven years before. Sudden onset of severe chest pain. Physical and x ray evidence of acute pulmonary infarct. No change in electrocardiogram. Sudden death while convalescing from pulmonary infarct. Without autopsy would have been ascribed to coronary occlusion. Autopsy showed no significant coronary sclerosis, no myocardial infarct. Death due to embolism of pulmonary artery secondary to static venous thrombosis. Clinical absence of heart disease more important than suggestive electrocardiogram abnormalities. (e) Male aged fifty nine. Attacks of nausea and vomiting, pain down both arms and into neck. After six months rest from hard manual labor, no symptoms. Good cardiac effort response. Lead IV raises question of old infarct in view of history, but because of absence of symptoms and other findings should now be allowed and encouraged to find sedentary work. (f) Male aged fifty seven. Hypertension. Attacks of chest pain, some of long duration. Electrocardiogram raises question of myocardial infarction. Serial records did not show progressive changes and showed only slight changes over a five year period. Uremic death. Autopsy—no sclerosis of main coronary stems, no gross myocardial infarction. Marked arteriosclerosis with narrowing of lumina and marked diffuse myocardial fibrosis and fragmentation. (g) Female aged twenty three. No complaints. Routine electrocardiogram showing abnormalities frequently attributed to coronary disease. No history or physical findings suggestive of cardiac damage. Excellent cardiac effort response. Clinically a normal heart. Electrocardiogram should be disregarded. Patient followed five years. No change in electrocardiogram. No symptoms of heart disease.

background, his pain threshold, his temperament, business environment and responsibilities, the amount of drive and energy he puts into his daily work, his ambitions and fears, his tendency to worry, his domestic environment, the demands on his time outside of business, his dietary habits, the manner in which he employs his leisure, and his philosophy of life. All too frequently there are factors of environment over which the patient has no control, yet which contribute materially to cardiac exhaustion. He may be caught in the stupid rush and drive of the American way of doing business which too often results in the premature retirement or invalidism of executives, just when their experience and mental capacity have become most valuable. It is unconstructive to dismiss the patient with the vague advice to "take it easy and not to worry." Invariably the patient is frightened by the thought of angina pectoris, whether he admits it to himself or not. From reading newspaper obituaries he associates the term with the possibility of sudden death. The wise clinician will avoid the use of the term "angina pectoris" even denying at times that such is the condition. Growing older, or aging, is such a gradual process that *its implications are lost to the average individual or they have only vague significance.* Business or domestic responsibilities are started in youth when bodily reserve is more than adequate to permit of excessive drive and insufficient rest without symptoms of overload. Habits of work thus formed are continued through the years without thought or understanding of the diminishing reserve. Hence the frequency with which we see men in their late fifties slapping their chests and proclaiming that they are "as good as they were thirty years ago." The early warning signs of breathlessness and easy fatigue go unheeded. When the attack of angina comes the mental reaction is a mixture of surprise, resentment, and fear, a blunt diagnosis of angina pectoris may lead to mental rout. By pointing out the analogy between this pain and the pain sometimes experienced by young athletes at the end of a race the physician can implant the idea that *any* heart, if overtaxed, may give warning of the overload by an attack of pain. Then calling attention to the patient's gray hairs and skin changes, he may be told that he cannot expect to have these outward manifestations of advancing years without also having some "gray hairs" in his myocardium. Logically, he should not expect his heart to be capable of the same amount of work at fifty or sixty as at twenty or thirty. By disregarding this fact and continuing the work habits of young manhood his heart has become tired and Nature has flashed a warning. If the warning is heeded and he cooperates in working out a sensible mode of life, he has many years of real usefulness ahead of him and he need not feel that a sword is continually hanging over his head. He must understand the problem.

In other

cooperation on his part. He must learn to do his work with the minimum expenditure of nervous and physical energy, that emotional outbursts or sudden marked changes in the level of physical activity are to be avoided, that his schedule should be so arranged that he does not have to rush from one thing to another, that having done his best in any situation, there is no use fretting over the outcome. In short he needs to develop a philosophical barrier between himself and the hustle and bustle of his environment. A private discussion of the problem with the wife or husband of the patient to

enlist cooperation in avoiding a too strenuous social program, to see that adequate hours of rest and sleep are obtained, to relieve the patient of as many of the domestic cares as possible, and to further the development of a hobby, is a valuable part of management. Actual business, economic, domestic, and social maladjustments or frustrations may not be entirely correctable, but the physician may be able to lessen their severity or teach the patient to compromise with the situation.

If hospitalization is impractical it is possible, of course, to carry out the analysis in the physician's office or laboratory with the patient remaining in bed at home between examinations. Sometimes it will be better to postpone detailed examinations until after a period of bed rest at home. If during the period of analysis and rest no further attacks occur, the patient may gradually be returned to a level of physical activity similar to that required by his business. If this is tolerated without symptoms he is permitted to return to work for a few hours, gradually increasing as the days go by. At all times he is instructed to stop whatever he is doing at the first suggestion of whatever his particular anginal equivalent may be.

By following the plan suggested above and sedative and other medication to be suggested later it will be found that many patients will go along, some for many years, without further attacks. If the attacks persist in spite of rest and all corrective measures and medication designed to improve the situation, or if on attempt to return to a reasonable day's work the attacks return with such frequency as to render working impossible, a vacation of several months or a year in a moderate climate may be followed by improvement sufficient to permit return to a sedentary occupation.

Active therapeutic measures consist in the removal or draining of accessible foci of infection, correction of intestinal stasis, correction of anemia, removal of a toxic thyroid if present, relief of eye strain, improvement of defective hearing if possible, correction of dietetic errors, reduction of overweight, and relief of hypertension if present. The importance of correcting even minor degrees of anemia is frequently overlooked. A hemoglobin content of 80 per cent with a red cell count of four million may be insignificant in a young person, but in an elderly patient with anginal attacks it may materially increase the likelihood of an attack. Periodic blood counts are a necessary part of management. The diet should contain adequate protein for weight requirements (not less than 1 gm per kilo of body weight), adequate mineral supply, should be free of "gas-producing" foods and condiments and should be divided into three or more approximately equal meals a day. A half hour of rest and relaxation after meals is desirable. While no vitamin deficiency in the diet may be apparent, it is well to insure adequate amounts, particularly of B complex and C, by supplemental oral administration. After some weeks this may be discontinued, to be resumed only if the patient does not seem to be doing so well. Reduction of overweight in the aging should be very gradual, approximately a pound per week, and only with due consideration for protein, mineral, and vitamin requirements. Hypertension, when present, is a possible cause of the anginal attacks. Management and treatment of both have much in common and may be favorably affected by the same routine. Detailed discussion of the treatment of hypertension appears elsewhere (see p 477).

Patients whose business affairs require travelling may experience dis-

oxygen concentrations of 80 to 100 per cent may be necessary,¹² at other times concentrations of 50 to 60 per cent may suffice. Of more importance is the possibility that daily inhalations of oxygen, say during a rest period before the evening meal, may lessen the frequency of attacks and possibly retard the degenerative process in the myocardium. Oxygen is also useful in controlling insomnia.

Waldman¹³ and others have reported on the use of *testosterone propionate* in angina pectoris. Beneficial effect is claimed in 33 to 100 per cent of cases by the various investigators. Lessened frequency and/or severity of attacks has been observed in addition to improved sense of well being. Intramuscular injections of 25 mg each are given twice weekly for eight weeks. If no improvement is noticed, further injections are not indicated. Some patients relapsed at varying intervals following the first course and were improved by subsequent courses. In our experience about 50 per cent of patients on whom it has been tried received some benefit. The mode of action is still conjectural. Shute¹⁴ has reported enthusiastically on the effect of *vitamin E* in doses of 100 to 200 mg orally three times a day. We have been unable to duplicate these results and were unable to find other favorable reports. *Thiouracil*, 0.4 gm daily, has been advocated by Raab.¹⁵ Di Palma and McGovern¹⁶ reported unfavorably on its use but conceded it might be indicated in patients with elevated metabolic rates or as a therapeutic test in selecting patients for thyroidectomy. More patients must be observed for longer periods before final evaluation can be made.

If syphilitic aortitis is found to be the cause of the anginal attacks, appropriate specific therapy outlined elsewhere should, of course, be instituted (see p. 428).

The interdiction of *tobacco* and *alcohol* in any form of heart disease has come to be expected by the laity. Inasmuch as there is experimental evidence to indicate that smoking causes constriction of the smaller arterioles and capillaries,¹⁷ there is some basis for prohibiting the use of tobacco, except in those patients who experience severe emotional and nervous distress on attempting to break the habit. The use of alcohol in moderation is apparently without harmful effect. In fact, the daily use of small amounts of alcohol is advised by some clinicians (see p. 455).

If, after rest, proper management, and appropriate medical treatment the attacks continue with such frequency or severity as to make work impossible, *surgical measures* may be considered. Of the various operations which have been used (total thyroidectomy, resection of sympathetic ganglia, paravertebral alcohol injection of sympathetic ganglia, cardio-omentopexy, and the attachment of a portion of the pectoral muscle to the epicardium), *paravertebral injection* seems the most practical. Painful stimuli from the heart apparently reach the spinal cord by way of the first five dorsal sympathetic ganglia. Injection of these ganglia with alcohol has resulted in complete, or almost complete, relief in as high as 63 per cent and great benefit in 26 per cent¹⁸ of patients treated. Local anesthesia is used and considerable skill is necessary to place the alcohol accurately. An unpleasant after-effect, for which the patient must be prepared, is an intercostal neuritis which may last for months. Only relief from a painful symptom is obtained. The underlying pathologic process in the heart is not affected and, as far as reported, the chance of subsequent sudden death is no greater or less than would be

the case if the attacks of pain were allowed to continue. When relief from pain is obtained it is usually permanent and the effect on the patient's morale is good. With the warning sign of myocardial exhaustion (pain) gone, however, the possibility of overexertion with subsequent cardiac failure may be increased. A more recent surgical approach which apparently has some favorable effect on the underlying pathologic anatomy is *pericorony neurectomy* combined with ligation of the great cardiac vein advocated by Fanteux.¹⁹ Further experience with this interesting technic is awaited with interest.

Almost every patient with angina pectoris will raise the question of exercise. To answer this requires as much individualization as other forms of management or treatment. In many cases some form of recreational exercise is desirable. The more violent sports, such as tennis, handball, and squash, obviously should be avoided. Walking, "setting-up exercises," and massage are safest, since the degree of effort required is easily regulated. Golf, bowling, and swimming are within the scope of some patients, provided the physician can determine whether the patient does them easily and is willing to limit the amount of participation to conform to his demonstrated cardiac capacity. When in doubt it is safer to limit exercise to walking, mild calisthenics, and massage. Advice as to sexual intercourse must also be individualized. The intense excitation involved is always a hazard.

In spite of all efforts at management or medical or surgical measures for relief, there will be some patients who continue to have attacks with sufficient frequency to make retirement necessary. Confronted with this necessity, the physician's duty is not discharged until he sees to it that the patient has some hobby to compensate for the sudden change from an active to an inactive life. In few of life's crises is the wise counsel of the physician so sorely needed as here. Men, particularly, are ill-prepared for the sudden cutting off of their life's work. Hobbies, particularly creative hobbies, can be developed after fifty, but not easily. As Stieglitz²⁰ has pointed out, the hobby need not be an expensive one. The amount of effort required can be adjusted to the patient's ability. Not infrequently a patient who is unable to sit at his office desk and conduct his business without frequent attacks of angina will be able to engage in light gardening, wood or metal working, or photography without distress. The difference apparently is in the amount of nervous tension attending the two occupations.

ACUTE MYOCARDIAL INFARCTION

Symptoms. Sudden severe precordial *pain* accompanied by shock, fall in blood pressure, and followed by fever and leukocytosis is the classical clinical picture of acute myocardial infarctions. This is also the picture erroneously referred to as acute coronary thrombosis or acute coronary occlusion. The reasons for discontinuing the use of these terms clinically have already been given.

The general consensus is that infarction occurs without reference to precipitating cause. In support of this is the fact that it occurs frequently when the patient is at emotional and physical rest or even asleep. Some patients are more prone to attack at rest than otherwise. Others suffer sudden

case Clinically, this may be of only academic interest. When one considers the slim margin between adequacy and inadequacy of blood flow which may exist in a myocardial area that finally becomes infarcted, he is impressed with the possibility that seemingly insignificant factors are potentially capable of upsetting this balance. Minor fluctuations in blood pressure or blood flow may be sufficient to set off the sequence of events leading to infarction. Such minor disturbances may occur at rest or as the result of some effort or excitement. Infarction has occurred while the patient was listening to an exciting radio program. It has been known to occur after surgical operations during which a fall in blood pressure took place.^{3 21} It has also resulted from the fall in blood pressure caused by hemorrhage, shock, and sudden rapid lowering of intra-abdominal pressure.^{11 22} Experiences such as these have led to the statement that infarction occurs without immediately precipitating external factors. Perhaps it would be more accurate to say that it may occur without readily recognizable precipitating cause. Whatever may be the explanation, the fact is that this ability of infarction to occur under any condition of physical activity or inactivity is an important differentiating point from angina pectoris. Vague premonitory distress in the chest may be experienced some days or even weeks before infarction.

Many variations in location, severity, radiation, and duration of the pain are encountered. Myocardial infarction without pain is not uncommon. Detailed illustrations of these variations would require too much space. Suffice to say that one should not be misled because the pain is not classical or is absent. When pain is present large and repeated doses of morphine may be required for relief. Nitrites are not effective and may even make the pain worse.

Nausea and vomiting are fairly common. When this occurs with the pain located in the epigastrium, a difficult diagnostic problem may present itself. Failure to perform necessary surgery on an "acute abdomen" is just as undesirable as to operate on a patient with acute myocardial infarction mistakenly diagnosed as an acute abdominal emergency. Patients with known or suspected coronary disease do have gallstones, peptic ulcers, and so on. Careful, painstaking, and unprejudiced observation will be rewarded by a minimum of errors.

Syncope, shock, cold clammy sweat, and ashen color (pale cyanosis) are common accompaniments at the onset of infarction. These are also variable features. Their presence is helpful, their absence does not preclude the possibility of infarction. Syncope may be the only symptom in a given case.

Fall in blood pressure is an almost constant finding. It usually occurs early in the attack but pressure may rise during the pain period, then later fall below the patient's average level. An occasional instance of postmortem proved infarction has been observed where the blood pressure did not fall at any time. If the patient's previous average level is unknown, it may be impossible to say whether a fall has occurred or not. For instance, a systolic level of 160 of itself is noncommittal, but if the previous average level was 200 it represents a significant fall. On the other hand, a systolic level of 90 or less is presumptive evidence that there has been a fall, even if the previous average level is unknown.

Fever, while usually present, may be slight. It may disappear within twenty-four to forty-eight hours and thus may be missed unless a careful

changes have disappeared. The presence of either Q_1 or Q_3 alone is not to be taken as pathognomonic evidence of old myocardial infarction, however, since it has been observed in records of hearts showing no infarction at autopsy (see Fig 75, d). Q_1T_1 records are usually associated with anterior, Q_3T_3 records with posterior, wall infarction. Instances in which this does not hold, as well as instances of proved infarction in which the Q-T changes are not clear cut (see Fig 75, b), or in which there are no changes, are encountered. Electrocardiographic abnormalities do not give a clue to the size of the infarct. Finally, preexisting abnormalities caused by varying degrees of bundle branch block, complete A-V block, preponderant ventricular strain, digitalis, and many other conditions may obscure the electrocardiographic signs of infarction. No discredit of the value or use of electrocardiograms in acute myocardial infarction is intended. Properly used and interpreted they are of great value and may be the principal factor in differential diagnosis. Even greater care must be exercised in the interpretation of possible "residuals" of old (healed) infarcts, such as Q deflections, RST depressions or elevations, and T inversions (see Fig 75, d, f, g). Here, particularly, the patient's history and what his heart is able to do are the important factors. If a careful history reveals nothing and if there are no physical or other signs of a damaged myocardium, an absolutely positive diagnosis of old infarction should not be made on the basis of electrocardiographic evidence alone (see Fig 75, g). The physician should at least keep his suspicions to himself and watch the patient without instilling fear. When the electrocardiogram is interpreted by another physician who does not know the patient, it is unwise for the patient to be apprised of graphic abnormalities when they constitute the only adverse finding. If in doubt, the physician may find some excuse to have the patient examined by a competent cardiologist. It must be admitted that occasionally a patient will be seen whose symptoms are vague, whose physical examination and electrocardiogram reveal nothing, yet who shortly thereafter develops acute myocardial infarction, which means coronary sclerosis was present at the time of examination. It is better for the physician to sustain such embarrassment occasionally than to be responsible for haunting fear in the greater number.

Prognosis. In any case of myocardial infarction a guarded prognosis should be given at the outset. If it can be established with reasonable certainty that the attack in question is the first one, the patient has about a 70 per cent chance of survival. Rosenbaum and Levine²⁵ analyzed the various symptoms and signs of infarction in relation to immediate prognosis. Their conclusions agree with our own experience, *i. e.*, that immediate mortality is higher in patients with severe shock, persistent dyspnea, severe cyanosis, persistently low blood pressure, higher levels of temperature, leukocytosis over 15,000, gallop rhythm, persistent arrhythmias or rapid pulse, congestive heart failure, the more severe electrocardiographic changes, and the older the patient. In addition, it has been our experience that patients who have repeated attacks of chest pain (acute coronary failure) after infarction have a poor outlook. Persistently small QRS complexes in all leads of the electrocardiogram is an unfavorable sign regardless of the other changes (see Fig 75, b). Preexisting angina induced by relatively slight effort would seem to indicate a more severe degree of myocardial and coronary damage, hence the immediate prognosis in such patients after infarction is not so good. The

same is true where severe hypertension has existed, because of the narrowing of the coronary arterioles. Electrocardiograms made before infarction, while very useful in diagnosis, are of uncertain value in immediate prognosis after infarction. Electrocardiographic evidence of previous myocardial damage may mean more severe preexisting coronary involvement than would otherwise be present. On the other hand, electrocardiograms with minimal abnormalities are frequently obtained from hearts with most extensive coronary lesions.

In summary it may be said that the milder the symptoms and signs the better the immediate prognosis. These criteria are only relative, however, since patients with the most unfavorable symptoms and signs do survive and some who appear the least disturbed by the attack die suddenly. The possibility of mural thrombi developing in the cardiac chambers is always present. Emboli may cause hemiplegia or other peripheral vascular "accidents" just when the patient appears to be doing well. Rupture of the heart at the site of infarction, with sudden death, may occur, but has been encountered only rarely in the autopsied cases personally seen.

Of those who survive a first infarction more than half are able to return to a sedentary occupation. Many of these lead useful, productive lives free of symptoms for years, if they live within the capacity of their hearts. Others have only occasional attacks of chest discomfort. The remainder experience all degrees of disability from partial, permitting a few hours of sedentary work a day, to total, permitting of little more than a vegetative existence. Only time and the response of the heart to gradually increased effort during a long convalescence can answer the question of prognosis after the acute stage. Even after the patient has returned to work and even though he may have few or no symptoms, and regardless of how long he may have survived his attack of infarction, there is always the possibility that another infarction or sudden death may occur at any time. It must always be borne in mind that myocardial infarction means serious interference with the circulation of at least two coronary branches. This is of more importance in determining the patient's future and outlook than the fact that a healed infarct is present in the myocardium. As is the case with immediate prognosis, only generalities can be used in ultimate prognosis after the acute stage. In our experience, most patients with persistently small QRS complexes in the electrocardiogram have lived less than a year regardless of other factors. Next in shorter duration of expectancy are those who experience frequent attacks of chest pain with or without obvious effort. While a patient may survive a second or third definite infarction, the level of existence and expectancy is lessened by each attack. Those with few or no symptoms on returning to sedentary occupations and particularly those without hypertension have the best expectancy and may live for fifteen years or more. The average expectancy of all those who survive a first attack is between five and ten years.

Treatment. *Rest, morphine and oxygen* constitute the essential therapeutic measures at the outset of an attack of infarction. The initial dose of morphine should rarely be less than $\frac{1}{2}$ grain, and subsequent administration sufficient to insure *absolute physical quiet* for the first few days is indicated. After the first dose of morphine, demerol 100 mg p r n may suffice to relieve pain and eliminate the constipating effect of morphine. For acute pulmonary edema, aminophylline, 0.2 to 0.4 gm intravenously, may be used. After the first

few days, sedation is required only as necessary to relieve pain, insomnia, or restlessness. *Both patient and family must understand that a period of absolute bed rest of six to eight weeks is imperative.* Oxygen given in high concentrations (60 to 100 per cent) relieves pain, dyspnea, pulmonary edema, cyanosis. With modern apparatus and service available, it is as readily administered in the home as in the hospital. Patients who receive oxygen do better both immediately and subsequently. Its daily use for thirty minutes to an hour throughout the convalescent period has appeared beneficial even though physical signs of oxygen want are lacking.

Papaverine, 100 mg every six hours, at first hypodermically and later orally, should be given from the outset of the attack. Mokotoff and Katz²⁶ have shown experimentally that vasodilators used from the outset reduce the size of the infarct. When convalescence is well established, the dosage may be reduced to 50 to 75 mg three to four times daily and continued indefinitely to promote the best possible coronary blood flow. For this latter purpose, the choice lies between papaverine and the xanthine derivatives just as in the management of angina pectoris. The routine use of *atropine* during the acute stages has been advocated²⁷ and is undoubtedly beneficial to many patients.

Since the danger of phlebothrombosis and embolism is always great in patients of the age group most likely to have acute myocardial infarction, *dicumarol* should be given from the outset if it is possible to do daily prothrombin time determinations. A dose of 300 mg the first day, 200 the second and 100 the third, will usually suffice to reduce prothrombin to 30 to 35 per cent of normal. It must be remembered that the effect of dicumarol is not felt for two to three and sometimes several days, and that there is considerable individual variation in response to the drug. When the desired reduction in prothrombin has been obtained, a daily dose of 50 to 100 mg will usually maintain the level. Each day's dose should be withheld until the prothrombin time has been determined and if blood prothrombin is below 30 per cent of normal, it should be omitted. If the prothrombin level falls below 20 per cent, 30 mg of synthetic vitamin K should be given intravenously daily until the level had returned to the desired figure.

Quinidine sulfate (0.2 to 0.4 gm every two hours) is useful if frequent premature ventricular contractions or paroxysms of ventricular tachycardia indicate the possibility of ventricular fibrillation. Auricular fibrillation complicates about 8 per cent of acute myocardial infarction.²⁸ If the onset is simultaneous with the infarction, quinidine sulfate 0.4 gm is given every two hours around the clock *except in cases showing conduction defects in the electrocardiogram.* In addition, *atropine sulfate* 1.3 mg is given intravenously followed by 0.5 mg orally three times a day. If auricular fibrillation occurs several days after infarction one may wait twenty-four hours to see if normal

and ephedrine
blood pressure
use of adrenalin
cerin is contra

indicated both at the outset and subsequently. We have not observed any appreciable benefit from *coramine*.

The diet should be light and if no food is taken for the first twenty-four to thirty-six hours no harm will be done. Easy action of the bowels without straining must be obtained, but frequent daily movements and purging are to be avoided. Abdominal distention is to be prevented rather than treated. If it should occur prompt measures for relief must be instituted.

It is desirable to obtain an electrocardiogram as soon as possible after the attack, both as an aid to diagnosis and as a guide to progress when compared with subsequent records taken first at twenty-four-to forty-eight hour intervals, later at seven-to ten day intervals. When there are no further changes from one week to the next, termination of the period of absolute bed rest may be considered, if everything else appears favorable. The first period out of bed should not be longer than twenty minutes of sitting in a chair at the bed side. If this does not cause a fall in blood pressure or a disproportionate increase in heart rate, the period of sitting up may be increased by five to fifteen minutes a day until two or three one-hour periods during the day are tolerated without fatigue or unfavorable effect on blood pressure or pulse. At this point a few steps may be taken around the room. At each increase in activity the physician should be on hand to check the effect, and the rate of increase of activity always kept under that which causes fatigue or signs of cardiac overload. By these gradual stages a level of activity comparable to that of a sedentary occupation may be reached. Six months is none too long in arriving at this point and a year may be necessary. The first effort at return to work should be limited to about two hours and the increase to a full day be gradually accomplished over several weeks. Even when a full day is resumed, fifteen-minute periods of relaxation at intervals during the day and a full hour for lunch and rest afterward are desirable. Ten hours of bed rest out of the twenty-four should be the permanent routine of life. For those able to afford it, daily periods of oxygen inhalation at home are beneficial.

Admittedly the above regimen is ideal and not always possible of application. Best results have been obtained where such routine could be carried out. Just as in the management of angina pectoris, an intimate knowledge of the patient plus attention to the factors of environment and regular observation by the physician are necessary for good results. The regular use of xanthine derivatives (aminophylline, glucophylline, etc.) or papaverine appears helpful. The remarks referable to mild anemia, adequate vitamin intake, etc., under angina pectoris apply equally to "postinfarction" patients. If, in spite of the best possible management, the patient experiences chest discomfort, codeine or morphine instead of nitroglycerin should be used. The resultant lowering of blood pressure following nitroglycerin is not without danger for it may precipitate another infarct. Sudden changes in the level of physical or emotional activity must be studiously avoided. Those patients who cannot be brought to a level of physical activity compatible with a sedentary job, or those whose training makes such a job impossible, must be considered totally disabled and encouraged to develop a hobby within their capacities.

Patients who are known to have old myocardial infarcts sometimes face necessary surgical operations (See Chapter 8.) Under such circumstances it

is of utmost importance to prevent a fall in blood pressure during and after the operation (See p 144) For this reason spinal anesthesia is not desirable unless acute congestive failure is present and surgery simply cannot be postponed If it is used the blood pressure level must be maintained by use of ephedrine and it would seem logical for the anesthetist to administer oxygen during the operation Dicumarol (see p 418) should be started on the third postoperative day to prevent phlebothrombosis These observations may well be extended to apply to any patient of middle age or over and certainly should be considered whenever there is a history of angina pectoris, hypertension, or where there is any reason to suspect coronary or myocardial disease Our anesthetic procedure of choice as worked out with Dr Mary Karp of Wesley Memorial Hospital is pentothal induction (about 200 mg) followed by ethylene and a very small amount of ether Oxygen should be started immediately postoperatively and continued for one to two days

ACUTE CORONARY FAILURE

Symptoms. Acute coronary failure is usually precipitated by some factor which increases the work load of a damaged heart or which, for the moment, impairs the efficiency of the heart Examples of the former are exertion, emotion, hypertensive crises, and aortic stenosis Cardiac efficiency may be basically impaired by coronary sclerosis, old coronary occlusions, old infarcts the occurrence of arrhythmias, tachycardias, heart block, congestive failure, shock, hemorrhage, trauma, infections, acute abdominal conditions, as well as acute pulmonary conditions such as embolism which may further depress cardiac efficiency to the point where acute coronary failure develops Some of these conditions may both put an added burden on the heart and decrease its efficiency Where the precipitating factor is exertion or emotion it is not always easy to say where the clinical picture of angina pectoris leaves off and that of acute coronary failure begins If one conceives of angina pectoris as the pain of a *damaged* heart *attempting* but failing to meet a work load, with the pain subsiding *promptly* as soon as the patient ceases the exertion, it will help in the differentiation The myocardial and coronary insufficiencies are transitory and recover quickly If, however, exertion is not promptly stopped or if the myocardium does not recover its efficiency quickly, the coronary insufficiency continues and the picture of acute coronary failure develops Other precipitating factors, such as those mentioned, tend, of themselves, to exert their effect over a longer period of time, cannot be stopped by the patient, and the resultant coronary insufficiency proceeds to the point of acute failure Thus, pain is apt to be of longer duration, resembling that of classical myocardial infarction

While a sudden fall in blood pressure may precipitate acute coronary failure, blood pressure changes caused by the failure are not so constant or

mon than in acute infarction An arrhythmia may precipitate acute coronary failure but the failure itself is not so likely to cause arrhythmia The incon-

stancy of the associated findings is confusing when one is attempting to make a differential diagnosis. If they are transitory the condition is likely one of acute coronary failure. The diagnosis may rest on electrocardiographic findings. When abnormalities are present they consist of depression of the RS T segment and inversion of T in one or more leads. Of more importance is the transitory and nonprogressive nature of the abnormalities, the record returning to normal as soon as the coronary failure ceases. Similar depression of the RS T segment and inversion of T may be observed within sixty seconds after compressing a main coronary stem in the dog. Upon release of compression the electrocardiographic abnormalities disappear within a very few minutes.

If an inadequate coronary flow continues beyond the acute stage because the precipitating factors persist, infarction may result either with or without thrombus formation. This is illustrated by infarction which follows surgical operations, hemorrhage, and shock. Here, a fall in blood pressure, especially if sudden, results in acute coronary failure. Infarction may occur almost immediately as the result of this failure or the patient may die suddenly before infarction has time to take place. In other cases infarction may not occur until some hours or days later. When this happens the lowered blood pressure will usually be observed to have persisted. Such infarctions resulting from acute or prolonged coronary failure may occur without frank coronary occlusion. That coronary thrombosis without infarction may occur under similar circumstances has also been demonstrated by postmortem examination, but there is no way of determining this clinically. It seems logical to assume that reflex lowering of blood pressure following intense emotional excitement, or the naturally lowered level of pressure during sleep, may be the precipitating factor in some attacks of acute coronary failure observed under these circumstances. It seems possible that such acute failure may, at times, result in gross infarction. The acute and often severe attacks of pain without effort, sometimes experienced by patients recovering or recovered from acute myocardial infarction, are understandable on this basis. The coronary circulation is already seriously impaired, and the blood pressure may not have recovered to an efficient level. That such attacks do occur without being an indication of fresh occlusion or infarction has already been

sible. Since coronary vascular disease and myocardial damage may be present without historical, physical, or electrocardiographic evidence, it may be emphasized again that a "negative" cardiovascular examination in a person past middle life does not justify failure to take all possible precautions to maintain adequate coronary flow in these conditions or during and after surgical operations.

Prognosis An attack of acute coronary failure should be considered as indicating some degree of coronary and myocardial damage just as in angina pectoris. The immediate prognosis is usually good if therapeutic measures are instituted promptly. Some permanent myocardial damage with possible lessening of efficiency will result if focal necrosis has occurred. Should the coronary circulation already be impaired by occlusions or marked

arterial narrowing, or the myocardium be severely damaged by fibrosis or old infarcts, death may occur suddenly without further occlusive or myocardial lesions

Treatment Prompt rest and sedation are paramount. Abnormal rhythms should be controlled by morphine or quinidine. Nitrites are of doubtful value and should not be used at all in the presence of definite or probably lowered blood pressure. Papaverine or the xanthine derivatives should be used as outlined in the preceding pages. When hemorrhage or shock is the precipitating factor, blood transfusion, or intravenous fluids, or human serum, and external heat are indicated. As in all cases of heart disease, intravenous fluid should not be administered faster than 1 liter in eight hours. Caffeine sodium benzoate is useful to support a failing circulation. If the blood pressure is low and does not recover promptly following these measures, adrenalin or ephedrine should be tried except where there is doubt as to whether infarction has occurred. For acute pulmonary edema, intravenous aminophylline given very slowly is preferable to atropine although atropine may be used if intravenous preparations of aminophylline or similar drugs are not at hand. Other agents of benefit in pulmonary edema are morphine, oxygen, venesection, and intravenous digitalis. For patients who have been on digitalis purpurea, lanatoside C, 0.4 to 0.8 mg intravenously may be used.²⁹

The period of bed rest depends on the degree of functional cardiac incompetence and the state of the patient's general health. If signs of focal necrosis (fever and leukocytosis) develop, a longer period of rest is indicated, but it need not, on this account alone, extend to the six to ten weeks usually desirable in gross infarction. In the definitely aged it may at times be better to cut the period of bed rest short to avoid pulmonary stasis or static venous thrombosis. (See discussion of dicumarol page 418.) While the patient is in bed detailed information concerning the patient and his cardiovascular renal system, as outlined in the discussion of angina pectoris, may be obtained. Subsequent management is similar to that of the patient with angina pectoris.

MEDICOLEGAL ASPECTS OF CORONARY VASCULAR DISEASE

The medicolegal aspects of coronary artery disease are of interest in connection with Workmen's Compensation Laws and double indemnity clauses in life insurance contracts.

In point is the case of a hod carrier who suffered an attack of chest pain as he lifted his hod to his shoulder. Subsequent events were typical of acute myocardial infarction. He stoutly maintained that he had not had a day off for illness in twenty-five years and had felt perfectly well until the attack referred to. Naturally, his days as a hod carrier were over. When his case came before the Industrial Commission the fact that he must have had coronary sclerosis for some time and could have had his infarction when off the job just as well as when on it was not the point at issue. The Commission wanted to know whether the effort of lifting the hod precipitated that particular infarction.

Other types of trauma such as falls, blows on the chest, and similar trauma are known to be followed by myocardial infarction, or acute coronary failure. Nonpenetrating injuries of the anterior chest wall have been followed

by small hemorrhages into the myocardium, usually without permanent functional damage if the heart is normal originally. This point may arise in connection with common carrier and public liability accidents as well as in industry. A workman of fifty or sixty may claim that attacks of angina pectoris, previously nonexistent, date from a nonpenetrating injury of the chest wall. A claim for double indemnity under an insurance policy may result from myocardial infarction alleged to be precipitated by an accident not of itself sufficient to cause death.

Opinions of clinicians of wide experience are not in agreement on the casual relationships involved. Court decisions usually favor the plaintiff or workman. Obviously each case must be decided on its own merits. It seems only fair to insist that, in order to be compensable, the infarction, or whatever, should result from effort extraordinary to the job and that symptoms and signs should follow such effort immediately or within a very few hours. The same criteria should apply to cardiac conditions alleged to be precipitated or made worse by accidents or chest injuries.

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CHAPTER 28

DISEASES OF THE AORTA

CHESTER S. KEEFER

THERE are three common diseases of the aorta in the aging and aged arteriosclerosis, syphilitic aortitis, and medionecrosis with dissecting aneurysm. All other disorders are rare. In this chapter these three diseases will be discussed, together with mycotic aneurysm of the aorta and infective aortitis without aneurysmal formation.

ARTERIOSCLEROSIS

In spite of the tremendous number of studies and investigations that have been made over the years, the cause of arteriosclerosis remains unknown. It is recognized that it is more common and extensive in the male than in the female, and it tends to occur at an earlier age period in men. It increases in frequency with advancing age and tends to involve all parts of the aorta, although the intensity of the process varies from one location to another. It is often most severe in the lower part of the abdominal aorta and extends into the iliac arteries. In the thoracic aorta, the process is most marked posteriorly about the orifices of the intercostal vessels, but the arch of the aorta also shares in the involvement. Individuals who live to be seventy-five years of age or older rarely show arteriosclerotic changes in the ascending arch of the aorta, although the abdominal aorta is often extensively involved by atheroma.

While the cause of arteriosclerosis is unknown, it is recognized that patients with diabetes mellitus, gallstones, or chronic nephritis develop the disease more often than others, and it is not infrequent in individuals with myxedema and xanthomatosis. There is a tendency for it to occur early in some families, and some races show a lower incidence of arteriosclerosis than do others.

Symptoms. Arteriosclerosis of the aorta, in itself, causes no symptoms unless the process involves the lumina of the larger blood vessels. If the process permits rupture to take place, or if the ulcerated area of arteriosclerotic plaques becomes covered with thrombi, then the aorta may become occluded or thrombi may become detached and cause embolization of its branches.

When arteriosclerosis of the aorta is present, the vessel elongates and loses its elasticity, it is therefore found to be very tortuous. In addition it often dilates, but a saccular aneurysm is rarely caused by arteriosclerosis. Most cases of aneurysm of the aorta which have been attributed to arteriosclerosis were described before very much was known about syphilis of the aorta, and since the two diseases often occur together in the same patient, it is unwise to attribute a saccular aneurysm to arteriosclerosis.

Diagnosis. An elongated aorta causes an elevation of the innominate artery so that it can be felt in the suprasternal notch. This elevation in turn

causes a kinking of the common carotid artery to the right of the sterno mastoid muscle on the right side so that it can often be palpated as an enlarged, dilated and kinked vessel. The only method of making a diagnosis of an elongated aorta clinically is by palpation of the elevated innominate artery in the suprasternal notch.

The dilated aorta on the right side may be detected by percussion of the border of the projecting aortic arch, and it may be suspected when there is visible pulsation in the second and third right interspaces of the chest wall.

The most accurate method of recognizing arteriosclerosis of the aorta is by means of *x ray examination* of the chest and abdomen. By this means, one can detect elongation and widening of the aortic arch as well as deposits of calcium in the wall. Before discussing the features of the dilated or elongated aorta it will be well to describe the appearance of the normal aorta.

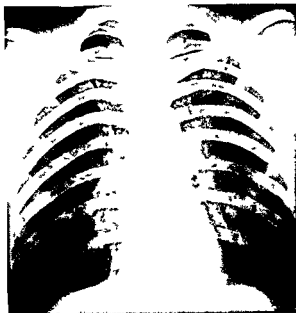


Fig 77 Normal aorta. Ascending arch is not visualized. Aortic knob is seen on left.

As age advances, there is a tendency for the aorta to lose its elasticity, so that it may elongate and dilate moderately. This may take place without extensive atherosclerosis of the wall of the aorta. When calcification is seen there, it is certain that the aorta is the seat of atherosclerosis.

As seen by x rays, the *normal aorta* has the following characteristics:

- 1 The supracardiac vascular shadow is always medial to the heart shadow on the right and it is not convex.
- 2 The right cardiac border is longer than the right aortic shadow.
- 3 The descending aorta bisects the heart at the auriculoventricular junction (Fig 77).

When the *aorta dilates*, the following features are noted:

- 1 The vascular shadow extends beyond the right border of the heart and is convex.

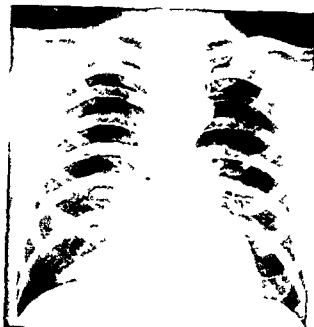


Fig 78 Elongated and dilated aorta showing convex border to the right and conspicuous aortic knob

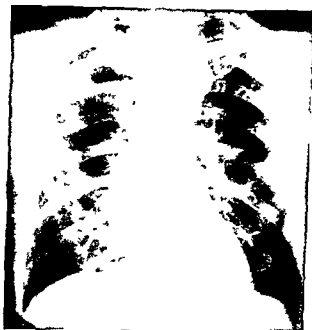


Fig 79 Elongated aorta extending to the suprasternal notch without conspicuous dilatation

2 The right cardiac border is shortened, owing to the posterior bulging of the aorta. This bulging displaces the heart downward, laterally, and to the left.

3 The arch of the aorta extends to the jugular notch and there is a widening of the vascular shadow above it (Fig 78)

When the *aorta elongates* it is recognized in the *anteroposterior view* by the following means

1 The length of the aorta from the level of the right cardiovascular junction to the level at which the aortic arch deviates medially is increased

2 There is an abnormal prominence of the aortic knob upward and to the left This is most striking when the aorta is dilated as well as elongated

3 The descending aorta is abnormally prominent to the left of the midline, and with extreme degrees of elongation its medial contour may be visible within the lung field Very rarely it may be seen extending to the right of the heart (Fig 79)



Fig 80 Calcification of the thoracic aorta without great dilatation

In the *oblique view of the chest*, the elongated aorta shows the following features

1 In the left anterior oblique view, the arch of the aorta shows a wide curve, there is overlapping of the vertebral shadow, and the aortic window is large The middle portion of the descending aorta swings far posteriorly and the lower part anteriorly, producing a marked curve or kink of the inner contour In some cases there is an indentation of the lateral wall

2 In the right anterior oblique view, the dilated aorta shows a steep course which converges toward the anterior chest wall and is almost parallel to the spine

When the descending aorta is displaced to the right, the trachea is displaced forward and the S-like shadow of the whole descending aorta is clearly visualized, its middle portion overlapping the spine, and its lower portion the retrocardiac space and dorsal aspect of the heart shadow 1

Changes in the *course of the esophagus* may also be made out. These have been studied with great care by Evans,² and are as follows:

1 There is an increase in the depth and elongation of the aortic arch impression on the esophagus by the aorta so that it has a ball and socket appearance.

2 Below the aortic arch impression the esophagus may follow the deviated aorta so that it is frequently seen to the left of the midline.

3 The esophagus crosses the aorta in the midthoracic region so that there is a deepening of the descending aorta impression.

Finally, when there is calcification of the wall of the thoracic or abdominal aorta as is seen in Figs. 80 and 81, there is no doubt about the existence of arteriosclerosis.



Fig. 81 Calcification of the abdominal aorta without great dilatation. Note the relative decalcification of the spine.

SYPHILIS OF THE AORTA

Next to arteriosclerosis, syphilitic aortitis is the commonest disease of the aorta. Since many cases are seen in patients more than fifty years of age, the two lesions are frequently present in the same individual.

In general, syphilitic aortitis causes a destruction of the media, proliferation of the intima, and fibrosis of the adventitia. These changes usually begin in the suprasigmoid region of the aorta, extend upward through the arch, to end at the beginning of the descending aorta. At times the process extends downward in the thoracic aorta, and stops abruptly at the diaphragm. In a few cases the abdominal aorta is involved, as are the sinuses of Valsalva. *The suprasigmoid area of the aorta is the region most frequently involved by syphilis.*

The anatomic lesions in the media cause weakening of the wall of the

aorta with dilatation and aneurysmal formation (fusiform or saccular), and owing to the intensity of the process in the suprasigmoid area, the aortic commissures are widened and aortic insufficiency results. The intimal proliferation causes partial or complete occlusion of the orifices of the vessels arising from the aorta.

The three commonest lesions, then, are aortic insufficiency, aneurysm of the aorta, and stenosis or occlusion of the ostia of vessels arising from the aorta.

One of the curious features of syphilitic aortitis is the long latent period between the initial infection and the development of clinical symptoms and signs. This is usually about fifteen to twenty years in length, so that most patients come under observation between the ages of forty and sixty. In some

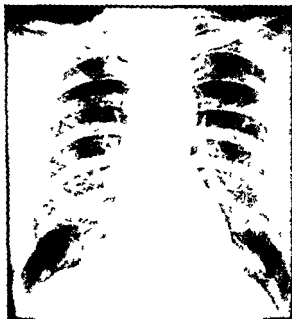


Fig. 82 Syphilitic aortitis with aortic insufficiency. General dilatation of the arch of the aorta without conspicuous lengthening.

series, at least half the patients have been less than forty-five, in others the average age has been between forty-five and fifty. The disease is much commoner in men than in women.

X-ray Diagnosis. There is nothing characteristic of syphilitic aortitis without aneurysm formation that permits one to make an etiologic diagnosis by x-ray examination alone. Dilatation of the ascending arch of the aorta with a convex prominence in a patient less than fifty years of age with syphilis, without hypertension, and increased pulsations limited to this area are highly suggestive of syphilitic aortitis. In a few cases there is a diffuse widening of the entire aortic shadow in the anteroposterior view, so that the supracardiac shadow is greatly widened. The left anterior oblique position often shows the dilated aortic arch, especially when there are deposits of calcium in the wall of the aorta. The right anterior oblique position shows a fusiform dilatation

with upward convergent borders if there are deposits in the aortic wall causing an increase in density (Fig 82)

AORTIC INSUFFICIENCY

Approximately 50 to 60 per cent of all patients who have syphilitic aortitis clinically develop aortic insufficiency, and about 10 to 15 per cent of these patients have an associated aneurysm of the aorta. The insufficiency of the aortic valves is due to widening of the aortic commissures, dilatation of the arch of the aorta, and scarring and retraction of the valve leaflets, with thickening and shortening. Thus, Von Glahn³ found by actual measurement that the circumference of the aortic ring was greater than normal in syphilis and that the free margin of the valves was shorter.

The diagnosis of aortic insufficiency usually causes no difficulty, since the local and peripheral signs are characteristic, but sometimes its etiologic diagnosis presents difficulties. In general, it can be said that between 90 and 95 per cent of all cases of aortic insufficiency are due to either syphilis or rheumatic fever. The other causes are arteriosclerosis of the aorta, bacterial endocarditis, and congenital bicuspid aortic valves with a superimposed process or an associated coarctation of the aorta.

Syphilitic aortic insufficiency most often occurs within fifteen to seventeen years after a primary infection in patients without a history of rheumatic fever. The aortic incompetence is free and there are peripheral signs of the disorder without evidence of other valvular disease. The aorta is dilated, and the serologic test for syphilis is positive in approximately 75 per cent of cases. The presence of associated signs of syphilis makes the diagnosis practically certain.

Rheumatic aortic insufficiency tends to occur in younger individuals and is more often associated with signs of aortic stenosis as well as mitral valvular disease, so that the regurgitation is less likely to be free. Reactions to the serological tests for syphilis are negative.

Arteriosclerotic aortic insufficiency is rare, and often there are only local signs of the disease without the peripheral signs. This diagnosis should be made with extreme caution and only after the other two common causes have been excluded.

ANEURYSM OF THE AORTA

Aneurysm of the aorta occurs in about 25 per cent of all cases of syphilitic aortitis. Since the anatomic lesion is most frequent and most intense in the arch of the aorta, this is the area most often showing an aneurysm. The aneurysm may be single or multiple, and it is about five times as common in men as in women. It is five times as common in the ascending and transverse arch of the aorta as in the descending arch, and while it is seen in the thoracic aorta, the incidence is about one thirtieth of that in the aortic arch. The

syphilis. All patients who are suspected of an aneurysm should be examined under the fluoroscope. The various features in the examination which are in favor of an aneurysm are as follows:

- 1 A history of syphilis and an associated aortic insufficiency
- 2 A lack of constitutional symptoms and relatively slow progress of the disorder
- 3 A pulsating mass
- 4 Displacement of the trachea and bronchi
- 5 Dilatation of the aorta
- 6 The rare occurrence of phrenic nerve paralysis
- 7 A negative response to x ray treatment

Aneurysm of Aortic Sinuses Small aneurysms of the sinus of Valsalva may remain latent or they may rupture into the pericardium, causing cardiac tamponade and death. Many of these aneurysms are associated with angina pectoris and aortic insufficiency, but they cannot be diagnosed during life since it is impossible to visualize them directly by roentgenography.

When these aneurysms become large enough to visualize, all of them except those arising from the superior arch of the aorta cause intracardiac bulging. A most careful study of these aneurysms has been made by Ostrum, Robinson, Nichols, and Widmann.⁴ The points they bring out are as follows:

- 1 *Aneurysms of the anterior or septal aortic sinus project through the sternum* and may be difficult to visualize in the anteroposterior view of the chest by x-rays. They are superimposed on the heart shadow. They can be visualized in the lateral view of the chest.

- 2 *Aneurysms of the anterior half of the right lateral sinus of Valsalva project to the right in the region of the right auricle.* This is the type of aneurysm that ruptures into, or interferes with the blood flow into, the superior vena cava or the right auricle.

- 3 *Aneurysms of the anterior half of the left lateral sinus of Valsalva encroach on the pulmonary orifice and cause obstruction to flow of blood through the pulmonary valve.* These aneurysms cause the signs of pulmonary stenosis, tend to erode the anterior parts of the second, third, and fourth ribs just left of the sternum, produce a pulsating shadow below the pulmonary area on the left, and may terminate by rupturing into the pulmonary artery.

- 4 *Aneurysm of the posterior half of the left lateral sinus produces signs at the upper left border of the heart above the pulmonary area and in the region of the second left interspace, and within the base of the left auricular appendage.* It compresses the pulmonary artery or may extend backward to involve the left main bronchus. It may perforate into the pericardium or erode the costosternal articulation of the second and third ribs.

All these aneurysms show silhouettes which, according to Ostrum and his colleagues⁴ are suggestive of a sac arising from one of the sinuses of Valsalva, and the diagnosis must depend on the roentgen ray findings together with the symptoms and physical signs.

Aneurysm of the Ascending Arch of the Aorta This is the commonest site for an aneurysm and the most frequent cause of a mass arising from the right side of the anterior mediastinum of vascular origin. The outstanding symptom is pain, which is localized over the front of the chest, usually to the right of the sternum. The *diagnosis* is made from the physical and x ray examination of the chest. The *physical signs* are those of a mediastinal mass (increased retromanubrial dullness), with increased pulsation and a prominence of the second and third interspaces on the right, with or without signs of compression of the superior vena cava (superior vena caval syndrome).

When these physical signs fail, the diagnosis can be made only by means of *x-ray* and *fluoroscopic examination* of the chest. The significant signs are a dilated ascending arch of the aorta with a convex prominence extending outward beyond the right auricular shadow, and increased pulsations limited to this area.

Oblique and lateral views of the chest are necessary in order to complete the x-ray examination.

The main points in the *x-ray diagnosis* are as follows:

1 A round pulsating shadow with a convex border, arising at the cardio-aortic junction, extending lateralward, and projecting into the right lung field.

2 Displacement of the arch and knob of the aorta upward and to the left.



Fig. 83 Anteroposterior view of the chest of a patient with an aneurysm of the descending aorta. Note the displacement of the trachea to the right.

3 Displacement of the heart downward and to the left.

4 Elevation of the right main bronchus.

5 Displacement of the trachea to the left.

6 The esophagus remains in the midline. (It is only in the very large aneurysms of the ascending arch that the esophagus is displaced backward and to the left.)

A very large aneurysm may project into the left side of the chest in the region of the pulmonary conus. It can be detected by lateral views of the chest.

arch, so that they cannot be distinguished as aneurysms coming to this

• trans
ending

section of the aortic arch alone. Nevertheless, when the transverse arch is involved, there is an elevation of the innominate artery so that it can be felt in the suprasternal notch. These aneurysms extend to the right more than the left, and when they extend forward they erode the sternum. Posterior extension causes tracheal compression. Other structures which may be involved are the recurrent laryngeal nerves, the esophagus, and even the left bronchus. Thus the patient may have stridor, coughing attacks with recurrent episodes of tracheitis or bronchopneumonia, and pulmonary atelectasis. Hoarseness, dysphagia, and a tracheal tug are common. The trachea is displaced to the right or posteriorly. Aneurysms in this location rupture into the trachea, left bronchus, left pleural cavity, esophagus, pericardium, or even the right pleural cavity. From the physical and x ray signs, they can be localized accurately.

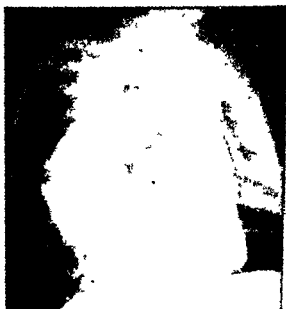


Fig. 84 Lateral view of the chest of the same patient as shown in Fig. 83, with an aneurysm of the descending aorta. Note that the aneurysm extends into the lower descending thoracic aorta.

Aneurysm of the Descending Arch of the Aorta. Compression of the left main bronchus and the esophagus with paralysis of the left recurrent laryngeal nerve is the commonest feature of an aneurysm in this area. Erosion of the spine from the third to the sixth dorsal vertebra or erosion of the ribs in the region of the scapula may occur. Rupture may take place into the left pleura or bronchus, esophagus, trachea, or left lung. It is in these cases, as in aneurysm of the transverse arch, that tracheal tug and bronchial and esophageal compression are so important in diagnosis, since a pulsating mass is usually not detected except by x ray examination (Figs. 83 and 84).

Aneurysm of the Thoracic Aorta. These are the least frequent of all the aortic aneurysms in the thorax, but when they do occur it is most often near the diaphragm. They compress the esophagus, erode the spine, invade the

pleural cavities or left lung, and pulsate in the left side of the back. The sac often projects beyond the border of the heart or displaces it. The pain from compression of the spine is often excruciating and the other symptoms most distressing.

X-ray examination of the chest in the oblique and lateral positions is essential, since in this view one may see the dilated aorta or erosion of the spine.

An aneurysm of this part of the aorta frequently presents itself as a fusiform or spindle shaped shadow to the left of the heart, or it may be completely covered by the heart shadow in the anteroposterior view. Sometimes it displaces the heart to the left. As a rule, the shadow is asymmetrical so that it is seen on one or the other side of the spine. Good lateral views of the chest with the esophagus filled with barium are important in diagnosis. When the aneurysm is in the midthorax, the esophagus is displaced anteriorly and to the right. When it lies just above the diaphragm the esophagus is displaced to the left. Other aids in the x-ray diagnosis are signs of dilatation of the ascending arch of the aorta, a widening of the aortic knob as determined by Kreuzfuch's method (that is, the width of an aneurysm is equal to or greater than the width of the ascending aorta), and erosion of the spine.

Aneurysm of the Abdominal Aorta. The abdominal aorta is the least common site for an aneurysm due to syphilis. Aneurysms resulting from arterio-

frequent than that of the posterior wall. The symptoms are due to interference with the blood supply to the intestines, compression of various structures, such as the spine, duodenum, and splenic vein, or rupture.

The commonest symptom is pain, which may be colicky or be accompanied by a feeling of fullness, pressure, and distention, obstipation, and vomiting. When the spine is involved (in 30 to 50 per cent of cases), the twelfth thoracic and first lumbar vertebrae are most often eroded, and pain in the back with radicular distribution is then frequent.

In some patients with arteriosclerosis of the aorta, there are no symptoms until rupture occurs. The *sign of an abdominal aneurysm* is a tumor which can be grasped and which has an expansile pulsation. The mass rarely moves with respiration, and a systolic murmur may be heard over it either anteriorly or posteriorly.

The most frequent mistakes are made in confusing a transmitted pulsation from the normal aorta through a tumor mass or a throbbing abdominal aorta. Examination in the knee chest position, x-ray examination of the abdominal aorta in the lateral position, and inability to grasp the tumor or to find that the pulsation is expansile are the essential points in diagnosis.

SYPHILITIC AORTITIS WITH ENCROACHMENT UPON OR OCCLUSION OF THE ORIFICES OF THE VESSELS ARISING FROM THE AORTA

One of the characteristic anatomic lesions in syphilitic aortitis is proliferation of the intima. When this occurs near the mouths of the various vessels

the openings are narrowed and in some cases may be completely occluded. Beyond the occlusion there may or may not be an organized thrombus.

The vessels which are most often involved in this process are the coronary arteries. Then, in order of frequency, come the left common carotid, the left subclavian, and the innominate artery, and the intercostal arteries. Only rarely are the orifices of the vessels narrowed in the abdominal aorta. Such narrowing reduces the blood supply to the various organs and in this way causes symptoms. When symptoms are not striking, there may be signs present which suggest that narrowing has occurred. These various features merit description.

Coronary Arteries Stenosis or occlusion of the mouths of the coronary arteries may be responsible for attacks of angina pectoris or sudden death. This anatomic lesion occurs in from 10 to 20 per cent of all cases of syphilitic aortitis seen at necropsy. Either the right or left coronary artery or both may be involved, and the lesion is most likely to occur when the coronary arteries arise above the sinuses of Valsalva.^{3 5} Stenosis is associated with aortic insufficiency in about 80 per cent of cases. The stenosis takes place so gradually that collateral circulation has time to develop and myocardial infarction is distinctly unusual. Of 120 cases of coronary artery stenosis collected by Von Glahn, myocardial infarction was found in only four. It is an extraordinary fact that a number of patients who have bilateral occlusion of the coronary arteries die suddenly without preceding symptoms.^{6 7 8}

Attacks of angina pectoris are commoner when aortic insufficiency is present. This is not surprising, since both lesions reduce the blood supply to the heart muscle (see p. 404).

The diagnosis of stenosis of the mouth of the coronary artery may be suspected ante mortem in any patient who has syphilis with aortic insufficiency and attacks of angina pectoris. It should be stressed, however, that angina pectoris may occur in aortic insufficiency without involvement of the coronary artery, or it may be seen in a few cases in which there is no aortic insufficiency. Stenosis of the coronary arteries may also be present in cases of sudden death.

Left Common Carotid Artery Stenosis of the mouth of the left common carotid artery is second in order of frequency. It may be involved alone or in association with the left subclavian or other arteries in the arch of the aorta. It produces decreased pulsation in the left carotid artery, and symptoms may occur if the closure is complete. These depend upon the rapidity of the closure, the condition of the cerebral vessels, the ability of the circulation to find collateral pathways, and the presence of complications such as thrombosis beyond the ostium of the vessel. When cerebral phenomena are present they consist of epileptiform seizures with attacks of unconsciousness, unilateral headache, and contralateral hemiplegia. Homolateral blindness with an associated optic atrophy may occur and pain in the neck and the upper part of the arm has been described. Encephalomalacia and cysts of the brain may be found post mortem, but the vast majority of these patients have no symptoms during life. It is important, then, that the carotid arteries be examined in every patient suspected of having syphilis of the aorta and in every patient with hemiplegia.

The whole question of carotid artery thrombosis has been discussed recently by Galdston, Govons *et al*.⁹

Left Subclavian Artery Stenosis or occlusion of the orifice of the left subclavian artery causes *pulsus differens* or complete absence of pulsation in the left brachial and radial arteries. There are often no symptoms. Pain in the arm with weakness and numbness may be a prominent feature after exercise of the limb and atrophy of the muscles has been reported. When the vascular stenosis is associated with an aneurysm of the aorta other signs of aortic disease are present.

Pulsus differens should always lead one to suspect disease of the aorta.

Innominate Artery Occlusion of this artery may be partial or complete and it may occur in association with occlusion of other vessels of the arch of the aorta or as an isolated lesion. It causes diminished or absent pulsation in the right carotid and right subclavian arteries. Symptoms may be entirely absent or the signs described under occlusion of the left common carotid and subclavian arteries may be present.

Intercostal Arteries Occlusion of these arteries is not uncommon in syphilitic aortitis and there are usually no symptoms associated with it. Cramps of the intercostal muscles during breathing and pain in the thoracic wall have been described by Oberndorfer¹⁰ and Gallavardin.¹¹

Mesenteric Arteries Obstruction of the mesenteric arteries due to syphilis is generally associated with aneurysm of the abdominal aorta but it may or may not be associated with thrombosis of the vessel itself. Recurrent attacks of colicky pain with abdominal distention, obstipation and vomiting are a feature of these cases. In one of my cases the inferior mesenteric artery was occluded by a thrombus which had partially recanalized. During life the patient had had recurrent attacks of severe abdominal pain. The involvement of the inferior mesenteric artery was associated with aneurysmal dilatation of the abdominal aorta and partial occlusion of the coronary and renal arteries. Death was sudden.

Occlusion of All the Vessels of the Arch of the Aorta These cases are most interesting. They have been described with and without an associated aneurysm of the aorta. The diagnosis is established by discovering no pulsation in any of the vessels of the neck or upper extremities. *These patients may have absolutely no symptoms due to the occlusion.*

When symptoms are present the patient complains of transitory dizziness especially when he changes from the recumbent to the erect position. Occlusion of the bilateral carotid artery has been associated in a few cases with atrophy of the facial muscles, premature cataract formation and glaucoma. Paralysis may not occur unless intracranial vessels are occluded.

OBSTRUCTION OF THE AORTA

Obstruction of the aorta is commonest in patients who develop thrombosis following an embolism of the aorta. The most frequent site is the bifurcation of the aorta at the origin of the common iliac vessels. The emboli in such cases generally arise from mural thrombi in the left auricular appendix or from the apex of the left ventricle or from thrombi forming on the aorta itself. When the embolism of the aorta is sudden it is followed by a characteristic picture. There is an abrupt onset of pain in the abdomen followed by a flaccid paralysis of the legs with numbness and sensory disturbances in them and diminution or loss of reflexes. There is diminished or absent pulsation in the leg vessels and pallor of the extremities. Gangrene of the

legs is rare unless an embolus becomes detached and enters one of the iliac vessels, or the thrombus extends for some distance into either iliac or femoral vessels. Complete recovery may take place, and there are cases where such an accident has occurred five years before death. This type of aortic obstruction occurs most frequently in heart disease unassociated with syphilis of the aorta.

On the other hand, the occlusion of the iliac vessels in patients with syphilitic disease of the aorta may be so gradual that very few symptoms arise. In these cases collateral circulation is well established, and in one patient whom I observed who had signs of occlusion of the aorta and syphilis, the collateral circulation was well established and large pulsating vessels could be seen on the lower abdominal wall. These vessels diminished greatly in size following antisyphilitic treatment. The only symptom referable to the lesion was occasional attacks of intermittent claudication. In the case recorded by Darling and Clark,¹² occlusion of both iliac vessels was complete and there was evidence of good collateral circulation.

RUPTURE OF THE AORTA

Rupture of the aorta may be due to a perforation of an aortic aneurysm, a dissecting aneurysm, or rupture through an arteriosclerotic plaque in the aorta. Rupture due to a syphilitic aneurysm or one causing a dissecting aneurysm occurs most often in the thoracic aorta, whereas that due to arteriosclerosis without aneurysm is most frequent in the abdominal aorta.

When a thoracic aortic aneurysm ruptures, hemorrhage usually takes place either into the pericardium or into the left pleural cavity, although it may pass into the esophagus, right pleura, left bronchus, trachea or even the mediastinum.

In the abdominal cavity, the rupture causes hemorrhage into the retroperitoneal tissues or the peritoneal cavity, the gastrointestinal tract, or even the thorax. The various pathways of dissection have been described by Jump and Leaman.¹³ Since there are certain important clinical features associated with rupture of the aorta they merit discussion in accordance with their etiology.

DISSECTING ANEURYSM OF THE AORTA

This is a disease of the aging, since most cases are seen in patients between the ages of fifty and seventy, and the essential lesion is a degeneration of the media of the aorta, for medial necrosis or atherosclerosis has been described in most cases. The rupture usually occurs in either one of two areas in the ascending aorta near the semilunar valves, or in the descending thoracic aorta below the origin of the left subclavian artery. Rupture of the abdominal aorta is much less frequent, unless the dissection begins there. From the site of the rupture in the intima, the dissection may extend along the course of the aorta and involve any or all of its branches, including the innominate, left common carotid, subclavian, intercostal, iliac, and femoral arteries. It may rupture externally into the pericardium, the pleural cavity (usually the left), or the mediastinum.

In a word, then, when the dissection occurs it produces *symptoms and signs* of acute blood loss and cardiac tamponade, increased mediastinal pressure, or interference with the blood supply to areas served by the various branches of the aorta.

Dissecting aneurysm of the aorta is five times as common in men as in women, and it is seen most often in patients with hypertension, chronic nephritis, or coarctation of the aorta. It has been described in epileptics and following trauma or exertion. Not uncommonly its onset is unassociated with exertion. The *diagnosis* is not always easy, since in many respects there is a close similarity between these cases and those of coronary occlusion (see p 412)

TABLE 10
PHYSICAL SIGNS OF DISSECTING ANEURYSM OF THE AORTA
Increased Mediastinal Pressure

<i>Organ</i>	<i>Symptoms and Signs</i>
Esophagus	Dysphagia
Superior vena cava	Engorgement of neck veins
	Edema of arms
	Swelling of face
Trachea	Dyspnea
	Suffocation
Interference with Blood Flow through Vessels	
<i>Artery</i>	
Innominate	Hemiplegia
Right carotid	Pulsus differens
Subclavian	Paralysis of arm
	Paraesthesia
Intercostals	Paraplegia
Superior mesenteric	Infarct of intestine
Renal	Hematuria
	Hemoglobinuria
Iliac and femoral	Absence or difference in pulsations
	Paresis of legs
	Gangrene

Displacement or Distortion of Aortic Valve

Aortic insufficiency

Rupture of Aneurysm

<i>Site</i>	
Pericardium	Pericardial friction rub
Lung	Hemoptysis
Left pleural cavity	Pleural effusion
Mediastinum	Increased mediastinal pressure

Clinically, the cases may be divided into two groups

1 Those in which death occurs suddenly, as a result of hemorrhage into the pericardium or pleural cavity

2. Those in which the onset of the illness is sudden with severe pain and there are signs of collapse and prostration, but in which the patient lives long enough to show signs of either increased mediastinal pressure with interference of the blood flow in branches of the aorta, an aortic insufficiency, a mediastinal mass, or a pleural effusion or pericarditis

In group 1, sudden death is the outstanding feature, and according to Shennan¹⁴ occurs in about 25 per cent of the reported cases. The diagnosis is made by the pathologist. In group 2, the features are frequently so dis-

tinctive as to leave little doubt concerning the diagnosis. As a rule, an individual, usually a man more than fifty years of age, who has had no previous symptoms of cardiovascular disease, although it may be known that he has hypertension, is seized with a sudden intense pain in the thorax or the abdomen, or both. This is soon followed by signs of collapse and prostration, and syncope is not uncommon. The attack often comes on during some muscular effort, although it may be spontaneous. Examination often discloses signs of aortic insufficiency, increased mediastinal pressure, interference with blood flow through the branches of the aorta, or rupture into the pericardium or left pleural cavity. A summary of these physical signs is shown in Table 10.

If the patient survives several days or longer, fever and leukocytosis are frequent. The electrocardiogram is likely to be normal, and the blood pressure



Fig. 85 Anteroposterior view of the chest of a patient with dissecting aneurysm of the aorta. The supracardiac shadow is widened, the aortic arch dilated and the aneurysmal bulge projects into the left lung field. There is a localized collection of fluid in the left pleural cavity.

continues to be elevated if there has been a previous hypertension. A normal electrocardiogram and the maintenance of the hypertension are points in favor of dissecting aneurysm of the aorta rather than coronary occlusion.

In patients who survive the acute episode, x-ray examination of the chest, as described by Wood, Pendergrass, and Ostrum¹⁵ is of assistance in diagnosis. The features as described by these physicians are as follows:

1. A deformity of the supracardiac shadow which is characterized by an arcuate excrescence extending beyond the wall of the aortic arch. There may or may not be pulsation.

2. A shadow produced by an extension of the dissection along one or more of the large branches of the aortic arch.

3. Displacement of the esophagus and trachea.

- 4 Left sided pleural effusion
- 5 Cardiac enlargement
- 6 Widening of the mediastinum

Rupture of Aortic Aneurysm—Thoracic An aortic aneurysm perforates in about 40 per cent of cases. When it does so it may cause rapid death, or death may be delayed for some days or weeks. Rupture takes place most commonly into the pericardium, the left pleural cavity, the esophagus, the right pleural cavity, or the left bronchus or trachea. Sudden or rapid death may follow hemorrhage into these areas, but it is most frequent following bleeding into the pericardium, esophagus, or bronchus. Death may be delayed when there is hemorrhage into the pleural cavities, the lung, the mediastinum, and even rarely the pericardium. In such cases the signs are



Fig. 86 Lateral view of the same chest as shown in Fig. 85 showing irregularity and widening of the descending aorta

those of a pleural effusion, a pericarditis, a mediastinal tumor, or recurrent hemoptysis.

Rupture of an Abdominal Aneurysm and the Abdominal Aorta An arteriosclerotic abdominal aorta may rupture and cause a dissecting aneurysm or a hematoma outside the vessel wall. This may occur with or without any preceding aneurysmal dilatation of the aorta. A saccular aneurysm of the aorta due to syphilis may likewise rupture. A complete review of abdominal aortic aneurysm has recently been presented by Jump and Leaman,¹³ and the points of rupture are outlined by them as follows:

Retroperitoneal rupture perirenal hematoma, pelvic hematoma, pleural hematoma

INFECTIVE AORTITIS

Abscess or infection of the aorta develops as a complication of bacterial endocarditis (mycotic aneurysm) or as an independent lesion, as in gonococcal, streptococcal, or staphylococcal infections. In some cases the infection arises as a metastatic process from a focus other than the endocardium, or it may arise by direct extension of an infection from a neighboring organ (spine or lung) to the aorta. In many cases the infection of the aorta occurs during the course of a generalized sepsis and may cause no symptoms or signs referable to the aorta during life. In others, erosion of the aorta by the infection causes hemorrhage into the pericardium with the signs of pericarditis or cardiac tamponade, or it may distort the aortic valves and cause the signs of an aortic insufficiency. If it occurs in other parts of the aorta, it may cause hemorrhage into the pleura, lung, retroperitoneum, mesentery, or even the esophagus.

The cases of greatest interest are *mycotic aneurysms* of the aorta without endocarditis. For some unexplained reason they seem to occur most often in the ascending part of the aorta above the aortic valves. In this region they may cause no murmurs for several weeks or months, but in most cases an aortic diastolic murmur appears sooner or later and leads one to suspect a lesion of the aortic valves. In some cases the aortic valve leaflets are normal, in others they are attacked and destroyed. These aneurysms of the aorta without valvular disease are due more often to gonococcal infection than to any other one infection, although the case reported by Crane¹⁶ was caused by a gram positive coccus. Aside from distortion of the aortic valves these aneurysms may leak and cause pericarditis.

In the *diagnosis* of a mycotic aneurysm in this region Stengel and Wolferth¹⁷ have stressed the importance of a vigorous, transmitted, visible pulsation in the second right interspace and a very loud diastolic aortic murmur in an individual with signs of bacterial endocarditis. To this list should be added the appearance of an acute pericarditis.

The feature of particular interest in these cases is that they can occur without valvular disease, and they may be associated with fever without

endocardial disease

I have seen a case of an infective aneurysm of the iliac artery due to

tion tests

Each case of the *pericardium* may arise from diastinal tissues. In such cases it will be impossible to decide whether the infection is of bacterial, influenzal or other respiratory types, are most important in the history. All the cases have been collected recently by Mills and Pinner.¹⁸

The symptomatology depends on the nature of the primary disease and on the location of the aortic lesion. The symptoms are due to fever and the

effects of increased mediastinal pressure and extension of the process to other organs in the thorax Hemorrhage is the most frequent cause of death

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CHAPTER 29

ARTERIOSCLEROSIS

IRVING S. WRIGHT

THE position may be taken and rather well defended that all evidences of senility are secondary to the primary mechanism of arteriosclerosis, that the signs and symptoms resulting are merely accidental depending on the portion of the arterial tree which is badly enough affected to prevent adequate blood supply to a part of the body, and that certain organs, when thus thrown into a state of imbalance, produce not only local manifestations, but far-reaching general reactions of great importance. Just as Zinsser¹ was able to present facts demonstrating the great importance of infection in world history, one might well marshal important evidence to prove that nations have fallen because of the presence of cerebral arteriosclerosis in their leaders—and countless examples might be cited of warped management and inhibited progress in many institutions as a result of this factor in more than one cortex around the directors' table.

Specific examples of general reactions within an individual may be found in arteriosclerosis of the arteries of the pancreas, with the secondary development of diabetes mellitus and its various complications, or arteriosclerosis of the arterial tree of the kidneys with the development of secondary kidney disease, hypertension and the many results of this syndrome.

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develop, with the pain sharply localized to the small mass of muscle which becomes ischemic, due to the circulatory deficit developing on exercise. Collateral branches may rather quickly take over the burden and relieve the acute deficit, or the syndrome may become more widespread as larger branches are blocked. A similar example of local damage may be noted in the gangrene frequently found in the tip of a digit due to blocking of an artery or arteries supplying that digit.

Multiply these examples by all of the organs and tissues of the body and the kaleidoscopic variations of pathologic change secondary to arterio-

the organ involved, but rather as arteriosclerosis with the organ relegated to a secondary position. This places the syndrome as foremost, both from the standpoint of occurrence of disease and as a cause of death. It is essential to discard the conception that arteriosclerosis constitutes the etiology of heart, kidney, or other secondary syndromes; they represent merely incidents in the course of this widespread process, about the specific etiologic factors of which we know very little. Such information as is available will, however, be considered.

INCIDENCE

It has been estimated that there are at present at least twenty five million persons more than fifty years of age in the United States. Of these 60 per cent, or fifteen million, will die of some cardiovascular renal syndrome, as compared with 9 per cent who will die of cancer. A very large but as yet undetermined portion of that 60 per cent will die as a result of degenerative changes of the arteries with secondary changes as already described.

ETIOLOGIC FACTORS

A problem of this broad magnitude and specific individual seriousness demands more careful consideration than has hitherto been given to it by either the medical profession or the lay public. We have accepted a modification of Marchand's² definition of the condition as follows: arteriosclerosis is a diseased or degenerative condition of the artery wall with thickening of the inner layer, accumulation of lipid substances, and further degenerative changes after calcification and distortion of the arteries. The so called Monckeberg sclerosis is closely related to, if not actually a manifestation of, the same process, with calcification of the media in the muscular arteries of the periphery and hyaline transformation of the arterioles with lipid deposits.

An understanding of the mechanism of the process is essential to the solution of the problem. Yet we must frankly confess that we do not even understand the etiologic factors responsible for its development.

Age. In man there is a roughly parallel incidence of increasing arterio sclerosis with increasing age. There are, however, many instances of young persons under the age of forty years who show marked evidence of sclerotic changes. There were more than 800 deaths from coronary thrombosis among the members of the armed forces under forty years of age during the past war, which were almost without exception on an arteriosclerotic basis. Two instances have been reported of arteriosclerosis, or, as it may be classified, atherosclerosis in infants under three months of age. This illustrates the rapidity with which the process can advance if certain little understood conditions are favorable. In contrast, many individuals sixty five or seventy years old reveal surprisingly few signs of sclerosis on postmortem examination.

Progressive Chronologic Arterial Changes. Certain changes take place in the arteries with increasing age. Among these is the gradual diffuse distention due to progressive deterioration of the elastic tissue. This stretching process occurs in the longitudinal as well as the circular direction. From early youth there is a tendency on the part of the internal elastic membrane to split and this becomes more marked as the years progress. Arteries are under longitudinal tension early in life, but by the average age of forty stretching has taken place which relieves this tension and from then on overstretching occurs, resulting in tortuosity. This deterioration of the elastic tissue with age¹.

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marked with senescence

Winternitz and his coworkers³ have published studies which suggest that these changes may be secondary to minute hemorrhages from the vasa vasorum. This work has been challenged by several workers on the basis of the technic of study used.

The fact that there is not an exact parallelism between age and the development of sclerosis inclines us to the belief that age *per se* is not the cause of these changes, but that they are secondary to one or to many of the factors which may enter into the life of the individual during the passage of time. The more significant of these possibilities will be discussed.

Sex and Endocrine Factors. A study of groups of both sexes by Lake, Pratt, and Wright⁴ demonstrated that under identical working conditions arteriosclerosis appears in marked form approximately one decade earlier in males than it does in females, and, furthermore, that severe degrees of calcification of the arteries are relatively rare in females under seventy, whereas

diet, but would not protect male rabbits. Repeating these experiments on castrated females demonstrated that castration deprived the females of some substance which was a key to this protection. It therefore appears that the endocrine glands, especially those associated with the so-called "sex hormones," play an important part in the development of, or protection against, arteriosclerosis, and that this aspect of the problem is worthy of much more investigation. On the other hand, recent workers at Columbia have succeeded in producing atherosclerosis in the vessels of dogs, following a high cholesterol diet, by producing an abnormal state of thyroid activity.

Race and Climate. Studies of the effects of race and climate on the development of arteriosclerosis have thus far been unsatisfactory and inconclusive. Clinical observations have been incomplete by their very nature. The cause of death as recorded frequently ignores the

Nutrition. There is no doubt that diet is of primary importance in the maintenance of health. (1) *Quantity of food.* (2) *Quality of food.* (3) *Changes in the ratio of various constituents.* (4) *Over- or under-supply of total food intake or of any of the vital constituents, such as vitamins, proteins, fats, minerals, etc.* (5) *Changes in the ratio*

of peptic ulcer, indicate that they have no more evidence of arteriosclerosis, and often less, than the average members of their age group in the population. The author has studied many such cases. Perhaps the most notable one is found in a man now seventy-seven years of age who has had a peptic ulcer periodically active since he was twenty-one. Through the intervening fifty six years he has taken an average of two to three eggs a day, plus a quart of milk. The rest of his diet has also been fairly high in animal fat. At seventy seven he is carrying on in an artisan trade which requires some manual strength. He is extremely active, enjoys surf swimming, and has less evidence of arteriosclerotic changes, by far, than most men of his age. His case is mentioned merely as an example with the understanding that it is but a single instance of this lack of production of arteriosclerosis as a result of high cholesterol diet.

Studies have failed to demonstrate that *disturbed inorganic salt metabolism* is a factor in the development of arteriosclerosis. Hypervitaminosis D, it has been noted, produces the deposit of calcium plaques, but these lesions are not characteristic of human arteriosclerosis. In spite of clinical impressions, there exists no proof that *overnutrition* leads to arteriosclerosis in man. Remarkably soft arteries are frequently found in obese patients. Evidence is also lacking that *undernutrition* is ever directly responsible for arteriosclerosis. No increase in incidence has been reported among the peoples of Central Europe who suffered from marked malnutrition during World War I. Of course, the children of that period have not yet reached the usual age for the onset of sclerosis, so we must wait to see whether dietary deficiency in early childhood manifests itself in arteriosclerotic changes in early senescence.

Some physicians contend that an *abnormal ratio of various food substances*, especially in the form of a high protein intake, increases the incidence of arteriosclerosis, but conclusive evidence to substantiate this is lacking. Eskimos, living on an animal tissue diet, do not show an increased evidence of renal or vascular disease. Ruffer⁶ performed autopsies on over 800 Moham medan pilgrims and found arteriosclerosis to be as common as in Europeans. This is significant in that these individuals never take alcohol.

Fat has been considered a potential cause of arteriosclerotic changes but this has not been established in man by controlled observations. Joslin⁷ and others have felt that a factor in the premature development of arteriosclerosis in diabetes has been excessive fat in the diet, body tissues, and the blood. This is frequently elevated in diabetes, but it should be remembered that diabetes is a disease affecting the general metabolism and the roles of the various factors have not been fully evaluated. Evidence appears to be accumulating that the newer low fat, high carbohydrate diets have not favorably affected the incidence of arteriosclerosis in diabetes. Further observations will be necessary to establish this relationship fully. In nephrosis and certain types of nephritis a high blood cholesterol is found associated with early development of arteriosclerosis. In these diseases, as well as diabetes, it seems probable that the factors at work may be endogenous rather than dietary.

In addition to the above mentioned data dealing with Mohammedan pilgrims there is much rather conclusive evidence that *alcohol* is not responsible for the development of arteriosclerosis. The studies by Lake, Pratt and Wright⁴ show that neither alcohol nor tobacco influences the incidence of arteriosclerosis. Pathologic studies show no increased incidence in alcoholics.

as contrasted with nonalcoholics.⁸ In fact, some workers believe that alcohol may retard the development of sclerosis as it is a cholesterol solvent and hence tends to prevent its precipitation.^{9, 10}

Arteriosclerosis may *interfere* markedly with the *normal nutrition* of the body. Even with a normal intake of properly balanced foodstuffs these patients often lose weight, develop deficiency diseases, and the tissues may be seen to undergo marked atrophy. The explanation for this usually appears to be on the basis of faulty utilization. This interference may take place at the point of absorption, an intermediate metabolic distribution or storage point, or at the area of utilization, namely, the cell to be nourished. This cell suffers no matter where the interference occurs. In order to compensate, diets rich in proteins, vitamins, and fruit should be used.

Acute and Chronic Infections. The problem of the relation of infections to the development of arteriosclerosis can be summarized as follows. In spite of exhaustive surveys and experimental work, there is little evidence in favor of the idea that infections play an important part in the pathogenesis of arteriosclerosis.

Heredity. While much evidence has been advanced to show a marked hereditary tendency in the incidence of hypertension, and this by inference applied to arteriosclerosis,¹¹ the direct evidence applying to sclerosis is less conclusive. It does appear, however, that in certain families the symptoms and pathology of peripheral arteriosclerosis occur at a younger age group than in others.

In any such group one must consider the average age of these families as compared with others, the type of work they engage in, the worry and strain of their lives, their dietary habits, and other factors—in other words, the total environment of these individuals as compared with any groups taken as controls.

Worry, Exercise, Hard Work. These factors have long been considered as possible contributing causes to the development of arteriosclerosis.

It has been believed for some years that the sclerotic plaques are most often found at the site of greatest strain of the artery, that is, where bends occur, where branches are attached, or where the artery is affixed to a nearby bone. Recent studies tend to show that increased physical strain may precipitate arteriosclerotic changes at an early age (forty to forty nine years) but that it probably does not influence the total incidence in the older age groups.

Lead and Manganese. These have been considered capable of producing arteriosclerotic changes in man and examples of this syndrome in printers, painters, and plumbers are often cited. Recently, however, the question has been reopened and cannot be regarded as finally settled.

PATHOLOGY AND PATHOGENESIS

The Individual Lesion. The changes in the arteries with increasing age have been described. In studying the pathology of arteriosclerosis a considerable variety of lesions are found which appear to be stages of development of the same process. The atheromatous type of plaques seen in the aorta wall may be found in other large arteries and develop as small, round, grayish, or yellowish spots which begin to project on the inner surface of the blood vessel. These gradually fuse into irregular, large plaques projecting further into the

lumen The development of fibrous tissue makes many of these become milky white, while the fatty parts break down, becoming mushy. From this point either of two processes may take place. The lesion is sealed by the formation of a calcareous plate or the softening material breaks down, producing ragged ulcerative defects at the bottom of which necrotic material may remain. Thrombi may then form and cover these ulcers.

Arteries of the Extremities The arteries to different organs show variations in the manner of development of arteriosclerosis. Our interest in this discussion must be focused on the vessels of the extremities. As explained above there is successive splitting of the intima which in senility results in a marked thickening of this layer because of the secondary development of fibrous tissue. This thickening is irregular, resulting in the lumen becoming eccentrically located. Degeneration and necrosis involve the intima and media with the deposit of cholesterol crystals. Fibrosis involves the media and adventitia. The arteries then become calcified by the development of circular solid deposits of calcareous material at the more or less obliterated dividing line between intima and media—the “pipestem” effect. The final closure is usually due to thrombosis. The process in diabetic cases is essentially similar.²²

Vasa Vasorum In the studies of Winternitz, *et al.*,¹ especial emphasis has been laid on the importance of the mural vascular channels, the vasa vasorum, in this picture. It was noted that very frequently small hemorrhages, fresh and old, were found in the walls of the vessels associated with arteriosclerotic changes, that they frequently proximated the orifices of smaller arterial branches as in the case with sclerotic changes, and that in certain instances small vessels were associated with these hemorrhagic areas. Careful dissection and study has demonstrated fine networks of blood filled capillaries on the intimal surfaces of the vessels, especially in the region of sclerotic plaques. It has been demonstrated previously that vasa vasorum are present in the adventitia and the outer half of the media. Careful clearing methods reveal the relationship of these vessels, hemorrhages, and calcified plaques to be such as to make it probable that hemorrhage from these small vessels eventually may result in the arteriosclerotic plaques of arteriosclerosis.

Thrombus Formation and Vascular Canalization Why thrombi form readily in some instances of damage affecting the intima of certain vessels and not elsewhere, where the damage is greater, is as yet an unsolved problem. There may be a relationship to the coagulating elements of the blood or to certain coagulating factors associated with local cellular changes. The mechanism of thrombosis is as yet little understood. Superimposed upon arteriosclerotic changes which may not even approximate closing of the lumen of a vessel, thrombosis is the terminal fatal event in the lives of more people than any other mechanism. The gradual narrowing of a vessel over a long period of time may allow for adequate collateral circulation to be formed, but the sudden closure produced by a thrombosis may not allow for that and may produce an infarction in the area thus suddenly deprived of its blood supply. If this happens to be in the heart or brain, or certain other vital areas, death ensues, otherwise gangrene and other complications may result. Many workers are today engaged in attempting to determine the factors essential for the production of a thrombus under these conditions. It must be considered that a balance between the prothrombin and thrombosing factors and the antithrombins, including heparin, is always in a state of flux. So long

as the antithrombin factors are predominant, thrombosis does not take place, but, when the thrombosing tendencies predominate, thrombosis does take place

The organization of the thrombus within a vessel is characterized by the disintegration and digestion of the blood elements accompanied by growth into the clot of capillaries and fibroblasts as well as many mononuclear phagocytes. This process extends until dilated sinusoids connected with tenuous narrow branches canalize the clot, which in turn calcifies in other areas. This may result in rather bizarre formations of vascular channels incorporated as part of the main lumen of the vessel involved. These channels are frequently primarily extensions of the vasa vasorum of the vessel wall. Thrombus formation may be initiated at times by exudative processes.

It is of interest that relatively few of the vessels of the vasa vasorum have been demonstrated in the arteries of young people.

Factors in Minute Hemorrhages. Numerous precipitating factors for these minute hemorrhages may be mentioned: dietary deficiencies, especially of vitamin C, hemorrhagic diathesis of various types, increased capillary fragility of other types, including chemical poisoning, parasitic infection, and other causes.

Winternitz, *et al*, conclude 'that while hemorrhage and perhaps lesser excitations are not the only source of the materials that form atheroma they are potent contributing factors. Certainly the hemorrhagic necrosis of intimal tissue already laden with fat-filled cells, results in the most extensive coalescence of lipid materials, in which cholesterol crystals and the debris of cells are found—calcification of the lesions of the artery wall is not a process which differs from calcification elsewhere. The primary requisite, necrotic tissue, preferably rich in lipoids is found in abundance in the vessel wall."

Foam Cell Theory. Leary¹² disagrees with this theory and appears to have demonstrated that phagocytic cells of the liver (Kupffer) and the adrenals engulf esterified cholesterol, thus becoming foam cells which then migrate through the vascular system finally invading the intima of some artery. This invasion starts the process of atherosclerosis.

True and False Aneurysms in the Peripheral Arteries. The purely *dilated, saccular, and dissecting aneurysms* are rather commonly found in arteriosclerosis. *False aneurysms* are, in our experience, less correctly diagnosed. They may occur at any point, but usually are found where stress and strain are common as at the popliteal space. The sclerotic artery actually splits or erodes through a plaque with a resulting hemorrhage into the surrounding intracellular space. A clot quickly forms which packs in firmly, forming walls. These may be more than an inch in thickness and are made up of all the elements of an old thrombus.

Arteriolar Changes. These may be the dominant factor in the picture and may be histologically divided into three main types.¹³ *Intimal hyalinization*, the arteriolar counterpart of simple arteriosclerosis, this is more widespread and severe with advancing age. *Medial hypertrophy and degeneration* resemble changes following distention of any hollow muscular structure. The medial degeneration may be primary in some instances, but the medial hypertrophy is probably secondary to stress and strain, being present more frequently in hypertensives than in nonhypertensives (see p 468). *Intimal proliferation*, in the form of endothelial hyperplasia with increase in elastic tissue and sec-

ondary degenerative changes, is classified as endarteritis obliterans rather than arteriolar sclerosis

ARTERIOSCLEROSIS OBLITERANS OF THE EXTREMITIES

Arteriosclerosis must be regarded as a generalized disease with essentially the same type of process potentially taking place in any or all of the arteries at one time. While our discussion will, for the most part, be confined to the effects of this process on the extremities, it must be pointed out that occlusion of any arteries in the body interferes with the nutrition of the supplied tissues and hence initiates signs and symptoms which should aid in making the correct, but often overlooked, diagnosis.⁵¹

Signs and Symptoms. Atrophy of the skin and nails, the latter growing poorly and brittle, is often the first sign. Atrophy of the muscles often accompanies this syndrome with marked loss in circumference of the extremity and total weight of the individual.

A common syndrome which develops early is the combination of pallor on elevation and rubor followed by cyanosis when the affected extremity is dependent. The color depends on the blood contained in the minute vessels of the skin and subcutaneous vessels. With impaired circulation, the blood drains quickly into the deep veins when the extremity is elevated and is not replaced rapidly enough because of the inadequate arterial supply. The result is a gradually increasing pallor to the point where the flesh appears almost pale yellow in color. When the limb is dependent the blood stagnates in these vessels, not being moved along because of the lack of arterial pulse pressure and other factors, a rubor is first produced which slowly becomes cyanotic as the oxygen in the blood is utilized.

In our experience edema occurs rarely, if ever, as a result of arteriosclerosis alone.

The patients frequently complain that the foot or hand is cold and that it is very difficult to warm it. Actual studies of the surface temperature confirm this observation.

Accompanying the sensation of coolness, numbness and "pins and needles" are often mentioned by the patient. Occasionally the feeling of formication is also noted.

The arteries may be found to be tortuous where they can be observed or palpated, i.e., the temporal, radial, brachial arteries. On palpation they are firm and not easily compressible. Frequently they are hard, either uniformly or in scattered areas where calcification has taken place. It is hardly necessary to say that when occluded no pulsation can be felt.

Fatigue and weakness are common presenting symptoms of sclerosis. The usual complaint is that after walking a certain distance (e.g., one to six blocks or upstairs) the muscles of the legs or feet become unusually fatigued. This symptom increases until it becomes the pain of intermittent claudication.

By pain of intermittent claudication we mean the ability of the patient to walk only a limited distance, at which point a cramplike pain in the muscles of his lower extremity forces him to come to a dead stop. Following a few minutes of rest the patient can then go on for about the same distance before similar symptoms once more force him to stop. The pain most commonly occurs in the muscles of the arches, calves, or thighs, and is characterized by a feeling of dull aching fatigue or a sense of cramplike constriction in the

affected muscles. The severity of the claudication is frequently increased by cold weather. It is also made more severe by walking rapidly or uphill.

Whereas claudication pains in the calves usually indicate occlusion of the popliteal arteries, we have seen a number of patients with this location of typical claudication pain, and with the major vessels open down to and including the dorsalis pedis and posterior tibial arteries. We have explained this as indicating involvement of smaller branches supplying groups of calf muscles and this has been demonstrated by arteriographic studies.

Rest pains frequently occur in the calf and thigh muscles of arteriosclerotic patients. They are typically cramplike in nature and are believed to be due to a local ischemia which results from stagnation of the blood flow, with secondary utilization of the oxygen and the accumulation of toxic waste products in the tissues.

Occasionally a patient is seen who has terrific rest pains in the feet with no demonstrable lesions aside from atrophy of the skin and muscles. We have seen examples of this so severe that despite all present-day methods of treatment amputation was required.

In general, however, the pain of arteriosclerosis is apt to be less acute but rather of a dull, aching nature. It frequently accompanies the development of ulcers.

The ulcers of arteriosclerosis tend to occur about the toes, ankles, or anterior tibial surfaces. They are usually dry rather than moist in nature, unless secondarily infected, which is rather common. They extend rather slowly, are often undermined on the edges and may develop a black eschar type of base. They may cause remarkably little acute pain, unless infected. These lesions commonly follow some trauma such as occurs in paring corns or nails too closely or stubbing the toes. The damaged circulation will not properly care for even such a slight degree of trauma and an ulcer develops which may never heal. In a sense these ulcers represent the first stages of a gangrene, since they are perpetuated by an inadequate blood supply to the area.

Gangrene in arteriosclerosis may be limited to a small area or may be massive depending on the size of the vessel involved and the completeness of its obliteration. Major vessels such as the femoral arteries, may close, however, without the development of gangrene if the collateral arteries have taken over the burden of supplying the tissues to an adequate degree. When gangrene does develop it may begin as a small bluish spot commonly on one of the toes, and gradually spread eventually to involve the entire toe, foot, or leg, at other times, an entire toe may be involved at once due to occlusion of a supplying artery, or if a major vessel occlusion suddenly occurs an entire limb may turn bluish black at one time. These gangrenous areas frequently progress to a dry, mummified state unless infected. When mummified they tend toward self amputation. Frequently, surgery is necessary to complete the severing process. If evidences of toxicity begin to develop, amputation above the level of occlusion, if possible, should be done promptly. This is especially important in diabetic patients with arteriosclerotic gangrene.

The suddenness with which occlusion may occur in arteriosclerosis is not widely enough recognized. An artery may have sclerotic plaques along its walls for years without much interference with its function. Suddenly, however, a thrombus may form about a protruding portion of a plaque and

occlusion may take place within a very short time. Many such episodes have been incorrectly diagnosed as embolic phenomena.

Findings by Special Examinations In addition to careful history taking and observation based on the above findings, which will frequently be sufficient to establish the diagnosis, there are additional tests which will help to confirm this impression and to establish the amount of damage involving the circulatory tree.

Oscillometric Readings These are useful in determining the patency of major vessels and the level of their occlusion. The important point is that of maximum pulsation and the amount of pulsation at that point should be noted. When the major vessels are functioning, pulsation will be noted. Otherwise, slight or no pulsation will be noted. This is more reliable than palpation since such factors of error as pulsations originating in the examiner's fingers and aberrant locations of the vessels are eliminated. By moving the instrument up the calf or thigh the level of closure can be ascertained where palpation is obviously impossible. Readings should be made in a room in which the patient has been warm for one hour or more, since chilling may produce spasm of the vessels and diminished pulsation. (It is important not to overinterpret the results of these or any tests later described.) The average normal readings at various levels are as follows:

<i>Leg</i>		<i>Arm</i>	
Arch of foot	$\frac{1}{4}$ to $\frac{1}{2}$	Hand	$\frac{1}{2}$ to 2
Above ankle	1 to 3	Above wrist	1 to 3
Below knee	2 to 5	Above elbow	2 to 8
Above knee	2 to 10		

Oscillometric readings do not give us information about the collateral circulation, which may take over the burden of the major vessels quite satisfactorily when the latter are occluded. This we must determine by other methods outlined below.

Surface Temperature Studies It is often quite unnecessary to use a thermocouple to determine by the skin temperature that the circulation is impaired. A marked difference between the two extremities is significant when they have been exposed to the same environmental stimuli for the preceding hour or more. A difference, even when only a fraction of a degree centigrade, can be readily recognized by the examiner's hands. The dorsal surfaces of the examiner's fingers are especially sensitive for this test. In arteriosclerosis the cooler extremity has the most markedly impaired circulation. If one wishes to record the difference and actual levels, a thermocouple or radiant heat measuring instrument should be used.

Reflex Vasodilatation Tests These tests are based on the observation that if one part of the body is markedly warm, vasodilatation will take place in other parts, especially of the extremities. If this is done to normal patients the temperature of the part being tested can be elevated from 32.9° to 33.9° C (91° to 93° F). If the circulation to a given extremity is impaired markedly there may be no rise above room temperature.

Landis and Gibbons¹⁴ suggested immersing the uninvolved pair of extremities in hot water, 43.3° to 44.3° C (110° to 112° F) for twenty to forty minutes. Maddock¹⁵ suggested wrapping the patient in blankets and hot water bottles. The use of a heat pad on the abdomen or wrapping both

arms in a heat pad is satisfactory. Other methods, such as blocking of the properly selected nerves (e.g., posterior tibial nerve for the feet), spinal and general anesthesia, and other means have been used but are usually unnecessary except as a check on the simple methods in doubtful cases.

Recently a drug which acts as a block for the sympathetic nervous system, notably tetraethylammonium bromide, has been used for tests of this nature. At present the response produced has not been fully evaluated.

X-rays. Flat plates, especially by means of a soft tissue technic, are useful for determining the presence or absence of arteriosclerotic plaques. We cannot, however, tell by the degree of sclerosis noted whether there is occlusion of the vessel or not. Vessels showing the most characteristic "pipe stem" calcification have proved, at autopsy, to be patent throughout, and, conversely, vessels showing only a few scattered plaques have sometimes been occluded.

Arteriography. Perhaps the greatest amount of information regarding the state of patency of the collateral as well as the main trunk arteries can be obtained by the use of a radiopaque substance directly injected into the arteries and x-rayed while in these vessels. This must be classed as a useful procedure especially where detailed studies are being made and where proper facilities and well trained workers are available. It is not necessary for routine study in most cases of arteriosclerosis.

Histamine Flare Tests. Histamine flare tests have been advocated as a test of the circulation. Following the injection of histamine intradermally, a flare normally develops extending 1 to 2 cm. from the site of injection. If the circulation is impaired this flare does not develop satisfactorily.

Measured Work Tests. If a patient is made to walk a certain distance or up a flight of stairs at a fixed pace and observed for the onset of claudication pains and the point beyond which he cannot proceed, valuable information will be gained for comparison with future studies after treatment. Ergometers¹⁶ have been described which measure this work capacity more accurately.

TREATMENT

The attitude of hopelessness with which the treatment of arteriosclerosis obliterans was formerly regarded is slowly changing to one of a constructive active approach. The result of this change has been a reduction in the number of amputations, the healing of arteriosclerotic ulcers, and the reduction of disability and pain.

The following procedures are of sufficient importance to warrant discussion.

Rest. Rest of the part is of importance with the following qualifications, any extremity with an ulceration or early gangrene should be completely relieved of stress or strain, even standing or walking. Prolonged, complete rest is now recognized to have serious dangers associated with it, especially in older persons. These include a greater tendency to thrombosis because of stagnation of the blood, and a tendency toward other complications including passive congestion of the lungs. This must be borne in mind, therefore, in the treatment of these patients. When, in the treatment of arteriosclerosis obliterans, an extremity is placed at complete rest, we mean that the patient should not bear his weight upon it or have it in a dependent position for long periods of time. He should, however, be encouraged to move it about in the

bed if this is clinically possible. Furthermore, such patients should not be kept in bed for very long periods of time unless frequent movement is possible, or unless the patient is on an oscillating bed which will be described later in this chapter. If prolonged immobility is imperative, the use of anti-coagulant therapy must be considered. This will also be discussed in a later paragraph. When sclerosis is present, without serious trophic changes or ulceration, the extremity may usually be exercised up to the point of pain. It is inadvisable for the patient to force the part beyond that point.

Position The position in which the limb is placed is of considerable importance for if elevated too much the tissues may become ischemic, whereas if it is lowered too far it may become engorged with cyanotic blood, which may also interfere with proper tissue metabolism. The most satisfactory position is one in which the limb is lowered until the tip is about six inches below the heart level.¹⁷ If the limb is elevated and then lowered slowly to the level at which the superficial veins just fill, this will be found to be about six inches below the heart level and the correct position.

Exercise Complete rest may, by its very inactivity, result in gradual stagnation, improper tissue nutrition, and cramplike muscle pains. Perhaps the best known exercises are those described by Buerger,¹⁸ which consist in principle of the patient lying flat in bed, first elevating the leg, then lowering it over the edge of the bed and finally resting it on the bed. The timing should be varied so that the color changes of pallor on elevation and rubor on dependency are observed and yet not maintained for more than a few minutes.

Allen¹⁹ has described certain helpful foot exercises as follows. The legs are hung over the edge of the bed and the feet are exercised by flexing and extending the ankles, turning the feet inwards and outwards, spreading and closing the toes. Pain may prevent the use of these exercises.

Exercises of these types should be performed two or three times daily for as long as possible (up to twenty minutes) without undue fatigue or claudication pain. An improved color and an increased warmth commonly result.

Tobacco It has now been generally recognized that the use of tobacco in the presence of impaired circulation is inadvisable. It was emphasized by Silbert²⁰ and others that in thromboangitis obliterans, smoking was a markedly aggravating, if not indeed, the commonest etiologic factor. Most clinics subscribe to this theory, at least in reference to aggravation. Experimental studies by Maddock and Collier,²¹ Barker,²² Wright and Moffat,²³ and Lampson²⁴ established the fact that the smoking of tobacco usually produces diminution of the blood supply to an extremity, normal or otherwise by constriction of the peripheral arteries, as determined by thermocouple readings, capillary microscopy, and plethysmographic studies.

Thus far, the only established mechanism involved is the physiologic one of constriction of the small arteries and arterioles. For the arteriosclerotic patient with already impaired circulation this is sufficient to interdict its use. While spasm of the sclerotic vessels is not considered important, one must remember that the life of the tissues frequently depends on collateral vessels that these are usually not sclerotic, and that they may be constricted by smoking.²⁵

Alcohol In contrast, the action of alcohol is definitely vasodilating. In experiments performed in our laboratory²⁶ rises in temperature of the tips of the extremities following the ingestion of 60 to 90 cc (2 to 3 ounces) of whisky have been as great as 5° to 6.7° C (9° to 12° F) depending, of course, on the condition of the vessels and the control level preceding the experiments. The use of whisky or other spirituous liquors is therefore indicated in the treatment of arteriosclerosis. With impending or actual gangrene the patient should receive enough to keep the peripheral vessels as dilated as possible. Often the patient will be kept very slightly inebriated during the critical period. This may require 30 to 60 cc (1 to 2 ounces) every four hours or more frequently. This may be tapered down as the emergency subsides to 30 to 60 cc (1 to 2 ounces) once or twice a day, which is the dosage usually recommended in nonacute cases. Whisky may also control the pain in some cases of vascular disease more satisfactorily than morphine.

Unless there are definite contraindications, these patients should have some alcoholic beverage each day for the remainder of their lives.

Baths The proper use of baths constitutes a definite contribution to the treatment of arteriosclerosis. These may be grouped under several headings, as follows:

Contrast Baths A commonly used form of bath for this condition is the contrast bath. There are, however, several objections to this type of bath which have resulted in our abandoning its use. First, the best containers only reach the knees. The blockage may be above that level so that, while the contrast baths may produce different metabolic demands, the ability to respond may not be realized at the level of the stimulus. Second, when vessels already damaged, are forced into sudden vasospasm by the cold water, they may remain closed, complicating the picture considerably. Third, in our experience there is often severe pain during the cold phase, perhaps due to cramping or marked ischemia. We have, therefore, adopted the long-used sitz bath for this purpose.

Sitz Baths With the modifications we have used, the patient sits in a tub containing at least 12 inches of water at a temperature between 34.8° and 40.2° C (94° F) for a period of thirty to forty minutes at least once a day. This overcomes all of the objections raised above in reference to the contrast baths. The heat extends high enough for collateral arteries from the trunk and femoral arteries to be activated.

Whirlpool Baths Whirlpool baths, if available, may further stimulate the circulation, the motion of the water being helpful, especially in the presence of a chronic low grade ulceration. The temperature of the water should be about the same as with the sitz baths (see p. 170).

Soaks Wet dressings have largely been abandoned in our clinic because of their tendency to get cool even under the most favorable conditions. This produces vasoconstriction and thus does more harm than good—defeating their chief purpose—that of improving the circulation to the dying cells. In their place we use soaks of normal saline solution with the temperature between 34° and 36.5° C (93.2° to 97° F). Even while using the soaks great care must be exercised to be certain that the temperature does not fall to the point where chilling is taking place. This may be prevented by the frequent addition of small amounts of warm normal saline. These may be repeated

one to three times daily for from fifteen to thirty minutes. After each soak the foot is removed, dried carefully, and placed under a warm lamp cradle, thus preventing chilling. The object should be to allow proper drainage by softening and cleaning away crusts which tend to lock in infection, but once infection is under control, attempts should be steadily made to produce a dry lesion rather than a wet macerated one. Healing is more rapid and danger of infection is reduced in this condition when the lesion is dry.

Heat. It is probable that in the past heat has done far greater harm than good because of its improper use. Properly used it should attempt to reproduce normal temperature conditions. The normal surface temperature rarely exceeds 33.9° to 35° C (93° to 95° F). In order to achieve this and hence stimulate normal metabolic processes, the temperature of the surrounding environment should approximate that level. We therefore use thermostatically controlled heat cradles which keep the temperature between 32.4° and 35.7° C (92° to 96° F). * Occasionally a patient will be found on whom it is more comfortable to use a temperature several degrees lower than these. Under these circumstances it is considered sound to make this adjustment. By careful watching and the use of a thermometer an equal distance from the lamp to that of the extremity, it is possible to use ordinary light bulbs, but the factor of error is great and many of these cases have been aggravated by such overheating in the past. Increasing the metabolic demands beyond the capacity of the supplying arteries tends to increase gangrene. Heat controlled within these limits is the only safe form to use. The local use of heat lamps, diathermy, or short wave machines is to be condemned. The author has seen over seventy-five cases of severe ulceration or gangrene which appeared to be precipitated by these measures—seven patients lost their legs and two died not as a result of their arteriosclerosis, but rather of the results of this misapplied treatment.²⁷ These modalities may be applied across the abdomen or hips to produce reflex vasodilatation (see p. 167).

Tissue Extract. Numerous workers during the past years, including Frey and Kraut,²⁸ Wolfe,²⁹ and Barker and Roth,³⁰ and members of our own group, have been interested in the possibility of the use of tissue extracts in the treatment of vascular ischemia, especially that producing intermittent claudication. Animal studies have demonstrated vasodilating and epinephrine-antagonistic properties of certain of these preparations. Evidence has also been presented that the intermittent claudication distances of arteriosclerotic patients as measured by voluntary work-load studies, and also by electrical contraction studies, have apparently demonstrated that this distance can be prolonged following the use of pancreatic tissue extracts (depropanex). Other studies have failed to demonstrate conclusively any evidence of vasodilating properties or other tangible evidences of a favorable effect upon the circulation. It must, therefore, be stated that at present final conclusions cannot be drawn regarding the definite value of this type of preparation. It is suggested that after a period of standardization of treatment including freedom from smoking, the use of alcohol, and proper exercises, has resulted in a definite measured walking capacity, and that further progress does not seem to be occurring, injections of 3 cc intragluteally, three times a week, and later twice a week, of depropanex may then be tried to determine whether further improvement be obtained. It has appeared in numerous patients that this

* We have found those made by Valverde Laboratories, New York City, satisfactory

is the case, but it is difficult to be certain that further collateral circulation is not developing as a basis of the total new régime. It is suggested that not fewer than twenty injections be given if one wishes to evaluate in any way its effect upon patients. Some patients have apparently regressed when the depromanex was changed without their knowledge to a placebo injection. Further studies in the field of the tissue extracts have seemed to be justified.

Pressure-Suction Boot Treatment and Intermittent Venous Hyperemia. With the passage of time the pressure suction boot^{32, 33} has been established to be of little importance in the treatment of peripheral vascular diseases including arteriosclerosis obliterans. Relatively few vascular clinics today use it as standard treatment. The author has not used the pressure suction boot in the treatment of such cases during the past four or five years. In his experience its use has sometimes been detrimental and rarely, if ever, helpful. The same conclusions have been arrived at regarding intermittent venous

By means of this so-called oscillating bed the head and feet of the patient are alternately elevated and lowered, a complete cycle taking from 1 to 2½ minutes. The movement is smooth, the patient soon becomes accustomed to it, and we have patients who have now used these beds for as long as four years. The beds have been in operation from eight to twenty-four hours a day. The degree of the tilting and the speed of the cycle can be regulated within reasonable limits. The object is to exercise the vessels which are still able to function by producing rubor and pallor (engorgement and ischemia) in a manner similar to Buerger's exercises, but continuously and without fatigue to the patient. This process is gentle, involves no constricting bands, and appears to be theoretically sound, especially in the treatment of arteriosclerosis with or without gangrene. We usually use a thermostatically controlled cradle at a temperature of 34° to 35.7° C (94° to 96° F) as an adjuvant treatment. We have now studied about two hundred and fifty patients who have used these beds. Clinical impressions are often deceiving guides and experimental studies are difficult in this problem, but so far the workers in our clinic and in other clinics have been very favorably impressed. Ulcers have healed in certain patients after complete failure of the pressure-suction boot and intermittent venous hyperemia. Many patients can use this equipment who are made very uncomfortable by these other methods. Thus far the chief indications for its use appear to be advanced arteriosclerosis of the vessels of the legs with impending or actual early gangrene.

Progress in this type of case is slow, it is usually necessary to keep the patient on the bed for at least one month.

Drugs and Intravenous Injections. Numerous drugs have been used in the treatment of arteriosclerosis obliterans on the basis of their vasodilating and other qualities. The commonest of these may be roughly divided into

- 1 The nitrites and allied compounds
- 2 The xanthines and allied compounds
- 3 The choline compounds
- 4 Papaverine
- 5 Potassium iodide
- 6 Hypertonic saline and citrate solutions

- 7 Drugs designed to block the autonomic nervous system such as tetraethylammonium or bromide
- 8 The use of ether intravenously
- 9 The use of histidine and ascorbic acid

In summary, the value of these drugs as established by the experience in our own clinic and other clinics of which we have personal knowledge is as follows. The action of the nitrites is too fleeting, and the dilatation which they produce as it affects the vessels of the extremities is observed only with difficulty and usually cannot be noted at all. The action of theobromine, aminophylline, and other members of the xanthine groups has not been demonstrated to be of value in the treatment of peripheral arteriosclerosis obliterans. The action of the choline compounds has not been found to be of value in the treatment of arteriosclerosis.

Papaverine has been advocated for the relief of sudden occlusion and for its general vasodilating qualities. We have found that when given by mouth or intravenously in the usual dosage its vasodilator effects are highly unreliable and not nearly so marked as the effect of simple reflex heat, or of 2 ounces of whisky given by mouth. However, recently we have been studying its effect following intra arterial injection in cases of sudden arterial occlusion. Here the results have been striking and definitely favorable. For example, in patients where an embolus or a sudden arteriosclerotic occlusion has blocked the popliteal artery at the level of the knee, the injection of 1 grain (0.06 gm) of papaverine in approximately 15 cc of normal saline into the femoral artery has on numerous occasions been followed by the development of a marked increase in warmth and flushing of the skin extending distally over the knee and down into the leg, and in one instance, to the foot. This effect has been produced in legs which have been cold and blue for many hours or even days. One patient who had received no relief from large doses of opiates for some five weeks experienced his first complete relief of pain following this injection. It is felt, therefore, that this method of using papaverine may well be further studied and utilized. The dosage to date has been 1 grain (0.06 gm). We have repeated it at four to six hour intervals when this seemed necessary.

Long standing use of potassium iodide in the treatment of arteriosclerosis merits its mention at this point, although clinically we have found little to convince us of its actual value. Animal work does seem to demonstrate that in rabbits it will retard the actual laying down of cholesterol plaques and it has been in use empirically in medicine for many years.

The use of saline and citrate solutions has entertained a certain popularity during the past years, especially as related to thromboangitis obliterans. There are no satisfactory theoretical grounds for the use of these substances in the treatment of arteriosclerosis obliterans.

Recently several new substances designed to block the sympathetic nervous system have been utilized. One of the most important of these is *tetraethylammonium bromide*. It decreases surface temperature and a fall in intravenously, the patient must be collapse from the sudden drop of takes approximately one hour. We have used it, for the most part, clinically in terms of intramuscular injection where the action is less striking but the

risk of collapse is markedly reduced. While the evidence is somewhat encouraging to date, it is too early to evaluate finally the position of these drugs in the treatment of arteriosclerosis obliterans. Recently, also, the use of ether intravenously, and the use of histidine and ascorbic acid in combination, have been proposed by workers in this field. Their final evaluation in the treatment of arteriosclerosis obliterans remains to be established. Certain other workers have not thus far been able to confirm the encouraging results reported by the originators of these forms of treatment.

Use of Antiseptics in Arteriosclerosis In all cases involving impaired circulation the use of strong antiseptics is to be condemned. The antiseptic will not serve its intended purpose if, in addition to destroying invading organisms, it also destroys the very delicate tissue cells which are attempting to establish a healing process. The use of iodine, the mercurial antiseptics, the silver salts, the phenols, or any other similar preparations is therefore, contraindicated. Hot soaks in boric acid solution or normal saline are preferred. We have found some stimulating substances such as the chlorines (dilute) (azochloramid solution 1:1000 in triacetin), and balsam of Peru to be helpful. For clearing away old slough and purulent sinuses benzine has frequently been very satisfactory when other solutions have failed to help. Recently the use of powdered human red blood cells (lyocyte) applied directly over these wounds has become popular. It is believed that it encourages granulation tissue. The final evaluation of this form of therapy remains to be seen. Thus far the results have been encouraging but not conclusive.

Surgical Aspects of Arteriosclerosis Although it is not within the scope of this work to present in detail the techniques involved in such surgical procedures as may seem necessary in arteriosclerosis, certain principles of established importance will be outlined.

The use of wet dressings, hot soaks, and antiseptics has been discussed previously. To reemphasize—*unless infection is present gangrenous areas should be kept in a dried condition*. Hot soaks are usually preferable to wet dressings. They should be at a temperature of 34.4° to 37.8° C (94° to 100° F), and should be given once or twice a day for thirty to sixty minutes. No strong antiseptics should be used since the life of the epithelializing cells is the most important single factor in the majority of these cases.

Lumbar ganglionectomy has been enjoying an increase in popularity in the treatment of peripheral vascular diseases of the lower extremities. Some enthusiasts feel that it is of definite value in the treatment of arteriosclerosis obliterans with gangrene and marked pain. Most surgeons feel that this should not be done unless a lumbar block with novocain has demonstrated a definite vasodilating effect. Others feel that the operation should be performed in the absence of such an effect on the basis that at times good results have been noted in cases where lumbar novocain blocks have not been satisfactory. On the whole our experience with this form of therapy has been disappointing, especially when injections have failed to produce satisfactory evidence of vasodilatation. Again, however, final conclusions cannot be drawn and improvement in technique may add to the value of this procedure.

Peripheral sympathectomy by either crushing, alcohol injection, or section of the mixed nerves supplying the feet may be helpful for the relief of pain. For correct technique the reader is referred to Smithwick and White⁴⁹ or Laskey and Silbert.⁵⁰ Such radical treatment is rarely necessary and should not be

used in the upper extremities since motor fibers may be destroyed at the same time as the sensory fibers, and although this is not so vitally important when it involves small intrinsic muscles of the foot, it is serious when it involves the hand muscles. Unless the nerve trunk is severed the nerve fibers usually regrow in from four to six months. The procedure should be performed with the greatest care to minimize trauma to the tissues, since healing is sometimes difficult owing to the impaired circulation which may be a factor high above the level of the gangrene. It is necessary to know thoroughly the location of each nerve and the tissues supplied by it in order to accomplish the desired results. Even then, in our experience, the presence of persisting aberrant branches or nerve fibers may cause the pain to be inadequately relieved.

The site selected for amputation is vitally important for successful healing. The day of amputating slightly above the level of the gangrene, whether it be in the toe or the foot, just because the skin looks good there, is over. Too often the patient is left with a poorly healed wound, a non-healing ulcer, and a high mortality rate.

We do not amputate today until nature has done her best to demarcate the area involved, to localize any infection present, and to attempt self amputation. If self amputation has progressed sufficiently in a toe, a moderate amount of help by the surgeon will sometimes complete the job and lead to local healing. If infection fails to localize, the gangrene continues to spread, or there is a systemic reaction taking place as a result of septic absorption, amputation is the only recourse, but not until a thorough study is made to determine a level at which healing can be reasonably expected. For this we use modern methods instead of guesswork. *The oscillometer will usually tell us where the major vessels are occluded and in the absence of further data it is unsafe to operate below that level.* The use of arteriographic studies using diodrast or thorotrast (thorium dioxide sol) gives us specific information regarding blockage of the major vessels and the extension of the collaterals. At times we are encouraged to amputate lower than the level of major blockage if very extensive collateral circulation is seen to be present. The histamine flare test is helpful in studying the circulation of the skin. If this reaction is inadequate the skin will probably fail to heal. Using these techniques, we are now able to conserve the maximum amount of limb possible, to reduce the number of reamputations markedly, and, as a result, reduce the mortality appreciably. This represents an intelligent, conservative approach to the problem of amputation which at best always represents an admission of defeat—our inability to save the extremity for future usefulness.

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CHAPTER 30

HYPERTENSIVE ARTERIAL DISEASE AND HYPOTENSION

EDWARD J. STIEGLITZ

HYPERTENSIVE arterial disease is much more frequent and more significant among the senescent than in the senile. Although hypertension is by no means limited to the years of later maturity, the great majority of cases fall within the age period from forty to sixty-five.

It is difficult, if not impossible, precisely to estimate the incidence of hypertensive disease. Consensus of numerous statistical studies indicates its occurrence in over 10 per cent of all American males less than forty five, rising to over 50 per cent at ages over sixty. The prevalence of hypertension in the relatively young men examined by the Selective Service during the years 1942 and 1943¹ was 18.4 per thousand (16.7 for white males, 27.5 for Negroes). However, these men were examined under conditions of stress. A survey² of some 15,000 persons over forty years of age showed that if hypertension is defined as any tension exceeding 150 mm. Hg systolic, and/or 90 mm. diastolic, the incidence of hypertension is as follows:

	40-49	50-59	60-69	70-79
Men	$\frac{25.9}{}$	$\frac{40.6}{}$	$\frac{56.3}{}$	$\frac{65.5}{}$
Women	32.0	53.4	67.7	

Preliminary studies of the 1940 Census³ reveal that 19.7 per cent of the total population of the United States, or about twenty-six million persons, are between forty five and sixty five years of age. Taking the conservative figure of 20 per cent as the average incidence of hypertensive disease within this age period, it is evident that there are at least five million cases in the United States today. Adding the not inconsiderable number of those younger or older further swells this appalling total. It is significant that only a relatively small fraction of this multitude have knowledge of their hypertension, for the disease is asymptomatic until well advanced and is discovered early only when searched for. An even smaller group are receiving adequate medical attention.⁴

The mortality of hypertensive disease is obscure because the disorder contributes to several of the "causes of death" used in compilation of vital statistics. The factor with which it is directly recorded. Life insurance data show that the mortality rate is doubled or tripled for those with hypertension. The essential markedly⁵ thus the total direct and indirect mortality due to the disease is tremendous. The major direct causes of death are cardiac exhaus-

tion, cerebral hemorrhage, pneumonia, and renal decompensation, in the order named. Cardiac failure, due largely to exhaustion from the ever mounting burden of the rising peripheral resistance, accounts for more than half of the deaths.

It must not be inferred, however, that every hypertensive patient is in dire jeopardy. Early in the course of this insidious disease there is little immediate risk. Many persons survive many years despite marked elevation of the blood pressure. The disorder is persistently progressive, but usually the rate of progression is slow. In most instances the expectation of long periods of disability is of greater concern than the imminence of death. Such disability may range from minor limitation of permissible activities to the complete invalidism of the cardiac cripple or paralyzed apoplectic. Although established hypertensive disease does not regress, early institution of proper medical management may greatly prolong the interval between onset and invalidism.

Hypertensive disease possesses four characteristics worthy of special emphasis: (1) it is insidiously silent in onset, (2) it is progressive and self-perpetuating, (3) its etiology is often obscure because of the multiplicity of factors involved, and (4) although the pathologic and symptomatic consequences may vary greatly in different individuals, the genesis of all the changes is the same impairment of the blood supply to functioning tissue cells.⁶

THE NORMAL ARTERIAL TENSION

Two groups of factors affect the arterial tension in the absence of dis-

slight rise in systolic tension is not abnormal, but in those persons in more nearly optimum health the diastolic tension tends to fall with advancing age. It is not desirable for the diastolic tension to rise with age.⁷ Other factors affecting the normal range are sex, race, physique, and climatic environment. Prior to the climacteric the blood pressure of women averages about 2 mm Hg lower than in men of the same age. The effects of race are closely linked with those attributed to physique. In Asiatic and Oriental races the normal blood pressure is from 10 to 20 mm lower than in Caucasians, the former racial groups are of smaller and slighter stature. Residence in the tropics is associated with a lower average blood pressure, although hypertensive disease is not uncommon. Probably both the peripheral vasodilation necessary to control body temperature and the usual more leisurely tempo of life contribute to this reduction.

Sources of transient variations include emotional reactions, muscular work, pregnancy, posture, and changes in metabolic rate and in cardiac vigor. Emotional turmoil of any sort is the most significant of these factors.⁷ Fear, anxiety, anger, and annoying discomfort are particularly potent sources of transient hypertension. It is characteristic that the first readings of the tension of a new patient are as much as 20/10 mm higher than those observed at later consultations because of subtle apprehension and/or embarrassment at the time of the first visit. Psychosomatic factors are very significant in the etiology of hypertensive disease.⁸ Exaggeration of the rise in tension arising from emotional stimuli is evidence of unusual vasomotor lability and indicates vulnerability to the development of hypertensive disease. Such patients are potential

hypertensives.⁹ Such reactions are very useful clues for the prediction of future hazards when observed prior to established hypertension. However, one must

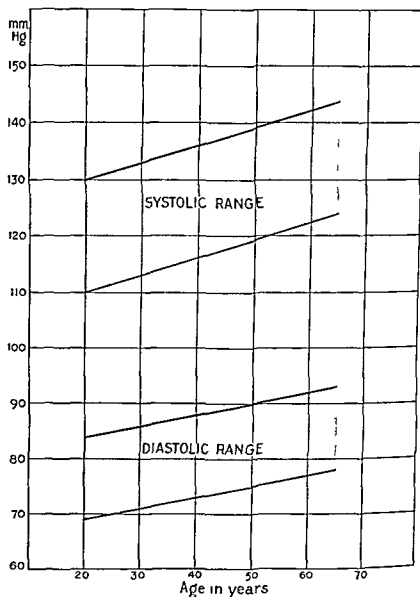


Fig 87 Range of normal arterial tension in men in relation to age. Data derived from hundreds of thousands of medical examinations for life insurance. Data beyond the age of sixty five are too incomplete for inclusion in the chart. Such information is much needed, not only in relation to the arterial tension, but in connection with all physiologic phenomena open to clinical mensuration.

be very cautious in drawing clinical conclusions from isolated observations of the arterial tension. Repeated observations on each individual patient are necessary.

THE MECHANISMS OF HYPERTENSION

The limitations of space preclude thorough discussion of the physiology of the circulation here ^{6 9 10 11} The forces involved in creating the intravascular pressure are (1) the propulsive force of the contracting heart and (2) the peripheral resistance

Propulsive Force The magnitude of cardiac work is rarely appreciated even by physicians The normal adult heart moves from 5 to 10 liters of blood per minute (125–200 cc per contraction) or about 10 tons of blood per day This weight of blood must be pushed into the aorta *against* the resistance of the diastolic pressure Thus a rise of diastolic tension from 70 to 105 mm increases the cardiac work at least 50 per cent It is far more surprising that the

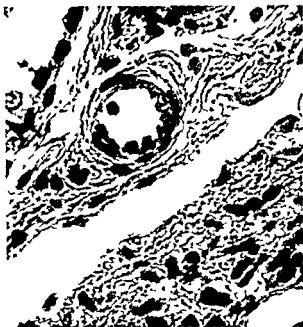


Fig 88 Arteriole magnified 735 times Phosphotungstic acid hematoxylin stain Lumen contains single lymphocyte Two rounded nuclei of endothelium bulge into lumen Elongated nuclei in circular smooth muscle fibers (Stieglitz *Arterial Hypertension* Paul B Hoeber Inc New York)

myocardial structures remain competent as long as they usually do than that they become exhausted prematurely in patients with hypertensive disease

Peripheral Resistance The caliber of the arterioles is the most important and variable factor determining the peripheral resistance The arterioles in addition to serving as conduction ducts in common with all blood vessels control the distribution of blood to various structures by localized dilatation or constriction and also maintain the pressure proximally by acting as sluice gates in a dam From 50 to 60 per cent of the fall in pressure from aortic to venous levels occurs in the arteriolar bed Constriction or relaxation is dependent upon the intensity of the flow of impulses along sympathetic nerve fibers to the arteriolar smooth muscle The vasomotor fibers are derived from the anterior spinal roots from the first thoracic to the fourth lumbar segment

The vessels of the upper extremities and the head and neck are innervated by fibers from the cervical cord. The whole vasomotor apparatus is partially controlled by a medullary center, with connections both through the spinal cord and the vagus nerve. The excitability of the arteriolar muscle fibers is under the influence of hormones, pituitary and adrenal secretions and probably other as yet unidentified chemical factors affect the muscular sensitivity.

Circulatory Equilibrium The circulatory apparatus is constantly in a state of kinetic equilibrium, adjusting itself to varying local demands for increased or diminished blood flow. Only extensive and generalized changes in arteriolar caliber are reflected as changes in blood pressure. With advancing years the lability and effectiveness of the homeostatic compensations depreciate, vascular dilation becomes less and less as the fibrotic changes of senescence affect the arteriolar walls.

Circulatory Imbalance and Hypertension Hypertension occurs whenever any generalized increase in arteriolar tone narrows the vascular bed. The increase in peripheral resistance is manifested by the rise in diastolic pressure. Elevation of the systolic tension is due to compensatory increase in cardiac force. *Hypertension does not involve new physiologic mechanisms.* The imbalance is due to quantitative rather than qualitative changes. A state of hypertension may arise from many sources: fear, anger, transient intoxications, endocrine imbalances (climacteric), and many others. If such a state of hypertension is brief, hypertensive disease need not necessarily arise. But prolongation of the primary arteriolar hypertonia, irrespective of the nature and sources of the continued stimulation, inevitably leads to hypertensive disease.⁹ With the single exception of the rare instances of aortic coarctation, with increased blood pressure in the upper extremities, hypertension invariably begins with increased arteriolar tonus. Conversely, hypotension is usually the result of undue arteriolar relaxation.

It is of the greatest importance to keep in mind the fact that arteriolar constriction not only raises the diastolic tension proximal to these vessels and thus greatly increases the cardiac load of work, but *also reduces the flow of blood distally into the capillaries.* Whereas arteriolar relaxation causes an active capillary hyperemia, arteriolar constriction may so retard the blood flow as to induce notable impairment of tissue nutrition and oxygenation. To describe this phenomenon David Riesman introduced the very useful term "histanoxia," which indicates that insufficient oxygen is being delivered to the tissue cells, although no true "anoxemia" exists. All the ill consequences of hypertensive disease are due primarily to histanoxia of essential parenchymatous structures. There is evidence indicating that one of the major biologic phenomena of senescence is a diminishing tolerance of aging tissue to reduced oxygen supply. Thus the consequences of arteriolar constriction (diastolic hypertension) are particularly menacing to those of advanced physiologic age. It is quite possible that premature senescence may be due to a combination of factors influencing cellular respiration.

HYPERTENSIVE ARTERIAL DISEASE

ETIOLOGY

Transition from the hypertensive state to hypertensive disease is insidious and gradual. A purely physiologic response becomes the major phenomenon of a progressive disease where hypertension persists for some time—and it

tends to persist. Although arteriolar constriction under appropriate stimulation is a purely physiologic reaction, it is characteristic that biologic reactions are in excess of requirements. Other compensatory reactions, such as fever, leukocytosis, edema, and the like tend to subside with the termination of stimulation. When the protective necessity for fever as a defense mechanism against infection is over, the body temperature falls. But hypertonic arterioles tend to remain spastic, although the original sources of stimulation have ceased to be operative. Thus a physiological response, which at first may well have been a protective mechanism, becomes a progressive disorder in which persistent arteriolar hypertonia leads ultimately to degenerative changes in the vessel walls.

Causative Factors The singular form of the words "etiology" and/or "cause" were best deleted from medical and scientific vocabularies. Nothing arises from a single cause. There are always several causative factors. These may be superimposed or occur in sequence, or both. There are three major categories of etiologic factors: (1) *predisposing*, (2) *provoking*, and (3) *perpetuating* influences. These groups may vary considerably in relative significance in different diseases, but they are all three invariably involved.

The key to understanding the complex etiology of hypertensive arterial disease is recognition of multiplicity in causative factors. The provoking etiology of hypertensive disease may be stated as being "anything which over a long period of time irritates (stimulates contraction of) the arterioles of a vulnerable individual." Such a definition requires amplification. It reveals three significant characteristics: (1) the importance of duration of irritation, (2) the multiplicity of possible irritants, and (3) the significance of predisposing vulnerability. It is important to remember that each and every case presents a new and individual diagnostic problem. The combinations of factors vary greatly in different instances of the disease. Although it is frequently impossible to do more than determine the *probable* causative factors, it is essential

arrive at such a conclusion. Intelligent and effective therapy requires understanding of cause. Diagnosis, which exists for the purpose of treatment, must therefore include study of etiology.

THE MORE SIGNIFICANT ETIOLOGIC FACTORS IN HYPERTENSIVE DISEASE

1 *Predisposing Factors* (determining individual susceptibility)

1.1 Heredity

1.2

2 *Provoking Factors*

2.1 Psychosomatic influences (anxiety, fear)

2 2 Intoxications

2 21 Exogenous

2 211 Metallic poisons (especially Pb, As, and Hg)

2 212 Habitual excesses of condiments

2 213 Habitual inadequacy of fluid intake (frequent)

2 22 Endogenous

2 221 Anemia (probably operative through histanoxia of nervous centers)

2 222 Fatigue intoxication

2 223 Pregnancy ¹²2 224 Renal disease,¹³ including { Nephritis
Hydronephrosis
Pyelonephrosis
(Apparently due to retention of pressor substance or substances associated with renal histanoxia)

2 23 Endocrine disorders

2 231 Gonad involution (climacterium)

2 232 Hyperepinephrinemia, medullary adrenal tumors (very rare)

2 233 Pituitary basophilism

2 234 Diabetes mellitus

2 3 Infections

2 31 Prolonged focal infections (frequent)

2 32 Generalized acute infections (especially influenza and typhoid fever)

2 4 Neurologic lesions increasing intracranial pressure (brain tumors)

3 *Perpetuating Factors* (often responsible for exacerbations)

3 1 Inherent in pathogenesis

3 2 Continuation of provoking factors (very common)

3 3 Superimposition of new provoking factors (frequent)

3 4 Impairment of renal and/or cerebral circulation by the hypertension

This outline is not complete. It is intended merely to suggest the type of clinical study indicated.

It is important to remember that the greater the predisposing vulnerability the lower the threshold to the provoking factors. Thus where there is a strong *family history* of hypertension, relatively minor insults may suffice to cause hypertension, whereas when there is evidence that hypertension is not a familial characteristic, one has reason to suspect rather intense and prolonged provoking irritation. The duration of irritation is often more significant than the intensity. An alveolar abscess neglected for years is to be suspected more than an acute but transient sinusitis.

The age at which the disease begins is another index to the patient's constitutional vulnerability. In general, the younger the age at the onset of hypertension, the greater the susceptibility or the more intense the provoking irritation. Exacerbations from various provocations are to be expected. In the management of hypertensive patients the physician must be alert to the menace of new sources of vascular injury.

PATHOGENESIS

Course. The pathogenesis of hypertensive disease follows an orderly course. It is not affected appreciably by variations in provoking etiology (Fig 89). Progression continues although the original initiating factors cease to be

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lation and thus arteriolar constriction is further enhanced. Prolongation of continuous spasticity causes fatigue of the muscle fibers. Muscle cells are not adapted to continuous contraction, but to intermittent work. Fatigue must not be confused with exhaustion, when response to stimulation diminishes or ceases altogether. Continuous constriction interferes with tissue nutrition and respiration, thereby encouraging the development of intimal fibrosis. As the hypertonia continues over a period of years, some of the arteriolar muscle

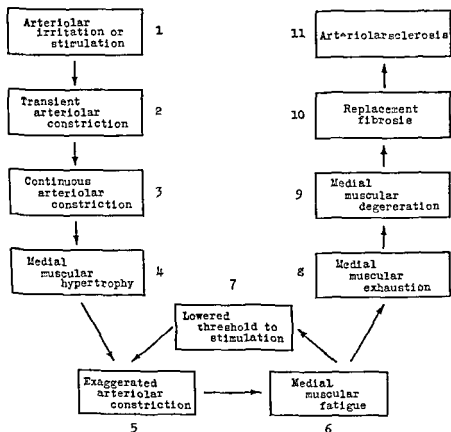


Fig. 89 Diagram of the pathogenesis of hypertensive arterial disease. Shaded frames represent the irrevocable anatomic changes and the vicious circle which is largely responsible for the perpetuation and progression of the disorder. Steps 1 to 7 are reversible and amenable to correction, steps 8 to 11 are irreversible and permanent (Stieglitz in Stroud: *Diagnosis and Treatment of Cardiovascular Disease*, F. A. Davis Co. and ⁶).

fibers become exhausted and die. Collagenic connective tissue replaces them. The insidious fibrosis of the arteriolar media is not an invasive or aggressive process, but a normal reparative response to parenchymal degeneration.

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emphasize the importance of a vicious circle in the progression of the disorder.¹⁵ The greater the constriction of the renal arteries the greater the renal

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ures are taken (see p 177) In the upper extremities contracture is flexor, in the lower extremities, extensor Apoplexy may recur several times, either in new areas of the brain or as an extension of earlier lesions The apoplectic lives in constant dread of such recurrences Tactful reassurance, frequent encouragement, and insistence upon occupation within capacity may do much to ameliorate these fears

Circulatory injury to the eye is frequent in hypertension Blurring of vision, scintillating sctomata, and amblyopia may be transient phenomena, retinal hemorrhage causes sudden and permanent unilateral effects An examination of the fundus should be an integral part of the clinical examination of all hypertensive patients for the retinal vessels are directly visible upon ophthalmoscopic inspection Valuable prognostic and diagnostic information is thus safely and easily acquired As a rule, the extent of retinal arterial change parallels the degree of cerebral arteriolar sclerosis (the retina is an integral part of the brain), but may be misleading as regards the stage of the disease in other parts of the body The rate of arterial degeneration is not uniform throughout the individual The retinal vessels may be more, or less, changed than those elsewhere (see p 294)

Cardiac Involvement. This is justifiably assumed in every instance of prolonged hypertension Three groups of causative factors are superimposed (1) myocardial anoxia (in common with other tissues), (2) ever increasing work, as the diastolic pressure mounts (the only structure so situated), and (3) probable myocardial injury from the original provoking etiologic factors, for the *myocardium is medial musculature* Coincident anemia or metabolic dysfunction (especially in glucose metabolism) seriously aggravates these cardiac insults Previous cardiac disease (see p 393) greatly lowers the margin of safety

Uncomplicated hypertensive heart disease goes through three stages (1) the sthenic stage, (2) the asthenic stage and (3) cardiac decompensation, unless some other catastrophe cuts life short before the final stage is reached The *sthenic phase* occurs early when the increased cardiac work leads to purely compensatory muscular development, prior to any impairment of myocardial nutrition and respiration During this stage the heart is not actually diseased It is vigorous and slightly hypertrophied, but not dilated The pulse is slow and no undue dyspnea follows exertion Electrocardiographic tracings reveal only a little left axis deviation and slight prolongation of systole (see p 364) The pulse pressure remains high

Transition into the *asthenic stage* proceeds by almost imperceptible degrees As the nutrition and oxygenation of the myocardium slowly becomes impaired, the compensatory response can no longer keep pace with the mounting load of left ventricular work Gradually the pulse becomes more rapid, dilation supersedes hypertrophy, and the margin for increased effort narrows Dyspnea is induced by less and less exertion The diminishing tolerance to exertion is often the only complaint at this stage, although precordial distress of varying intensity may be admitted Usually the patient's wishful thinking rationalizes the "short wind" as due to smoking, fatigue, lack of exercise, or,

degrees from the sthenic stage to frank *decompensation* (see p 386) It may persist for many years, or be relatively brief, depending upon the intensity of cardiac *histanoxia* and the cardiac status prior to the onset of hypertension Cardiac decompensation (p 385), angina pectoris (p 404), and coronary occlusion and myocardial infarction (p 412) are discussed elsewhere These three cardiac problems are not limited to hypertensive individuals, though the existence of hypertension invariably exaggerates and accelerates the cardiac damage

Renal Consequences In hypertensive disease the renal effects are reciprocal Ischemic renal tissue liberates a pressor substance or substances¹⁵ These noxae are removed by normal renal secretion The greater the renal anoxia, the greater the generation and/or liberation of the pressor mediator and thus the greater the renal arteriolar constriction and aggravation of the renal ischemia This vicious circle undoubtedly constitutes a potent perpetuating force contributing to the persistent progression of the disorder

Although clinically detectable impairment of the renal functions is the exception rather than the rule in hypertensive disease, renal function tests should be applied in the study of every case Such tests need involve neither much equipment nor expense Although no one test yields all the information desired,¹⁸ *functional impairment of the kidneys always involves both tubules and glomeruli* Glomerular injury impairs the blood supply to the tubules, tubular injury is hematogenous and the blood circulates through the glomeruli before reaching the tubules Thus functional impairment (and structural pathology) is always mixed, although the predominance of one or the other varies

The most physiologic and also the most sensitive test to detect early impairment is the stress procedure known as the "Concentration Test," whereby the renal work of concentrating the urine is increased by relative dehydration A most satisfactory routine is that proposed by Fishberg¹⁹ (See p 591) This procedure requires the minimum of apparatus, it should be a routine office test There is obviously no risk and a minimum of inconvenience for the patient Other desirable methods of studying the renal functional capacity are the Mosenthal concentration diuresis test, urea clearance, inulin clearance, and the phenolsulfonphthalein excretion test

Other Consequences Other effects of hypertensive disease include the phenomena of late diabetes mellitus (see p 216), gastrointestinal disturbances which often simulate cardiac disease, and circulatory difficulties in the extremities (see p 450) Let us not forget that the ill effects of arteriolar constriction are engendered by *histanoxia* of *parenchymatous structures* and that the symptoms are thus secondary to functional disturbances of various organs

PROGNOSIS

Individual Prognosis The average, or group, outlook for hypertensive pa-

and careful study are essential for prognostication, not only because of the many variable factors involved, but also because the program of management

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disease (requires knowledge of duration), (4) the stage reached in pathogenesis, (5) the character and extent of complications, including the status of the cardiac and renal reserves; and (6) the cooperation of the patient

The etiologic picture is important because certain etiologic factors are amenable to corrections; others are not. Fatigue, plumbism, dietary defects, anemia, obesity, focal infections and the like are open to therapeutic attack. Constitutional factors, long past intoxications and infections, nephritis, and intense occupational responsibility are far less amenable to therapy. The *duration* of provoking factors is significant. Unfortunately, etiologic diagnosis is almost always fragmentary and largely presumptive. Nevertheless every effort should be made to obtain all the information possible.

Advanced age affects the prognosis rather favorably. As a rule, the earlier in life the degenerative diseases begin the more rapid is the deterioration and the graver the outlook. This is well established for diabetes mellitus, gout, and cancer, as well as for hypertensive disease. True, normal life expectancy declines with aging. The outlook for a man of sixty-five with a blood pressure of 200/110 is distinctly better, however, than for his nephew, who at forty-six, already has a tension elevated to 170/105. The onset of hypertension at an early age implies either marked constitutional vulnerability or intensely active provocative arteriolar irritation.

The rate of progression of arteriolar changes can be estimated only if one knows the approximate date of onset and the stage of the pathogenesis reached. The former unknown is difficult to determine because of the silent and wholly asymptomatic early course. Painstaking inquiry into the past history, including examinations for life insurance and the like, will frequently reveal some clue as to the probable duration, however. Data from periodic health inventories are invaluable. The occurrence of arteriosclerosis within a few years of onset is proof of rapid progression and justifies a guarded prognosis. On the other hand, a statement such as "My blood pressure has been averaging around 170 for over ten years," warrants a much more optimistic prognostication.

Evaluation of the *stage reached in the pathogenesis* depends largely upon the variability of the diastolic tension. Direct inspection of the retinal arteries by retinoscopy gives additional valuable data. These data may be misleading, however, for the fibrotic scarring proceeds at different rates in different parts of the arteriolar vascular tree (p. 468). During the spastic stages (p. 470) the diastolic tension is labile. As fibrosis progresses, the diastolic tension becomes more and more fixed. Thus a rigid, moderately elevated diastolic pressure is more ominous than transient excessive diastolic hypertonia. Repeated observations over some weeks are necessary to reveal the degree of lability, unless one employs a vasodilator to measure the relaxability of the arterioles. By the "amyl nitrite test" one may quickly and safely find the answer to this important question.²⁰ The technic of this procedure follows.

Amyl Nitrite Test. With the patient comfortable either sitting or lying down, enough determinations of the arterial tension are made to obtain a fairly constant basal level. The patient then inhales twice of a freshly broken pearl of amyl nitrite (0.3 cc. or 5 minims). Rapid observations of the arterial tension are repeated until the ensuing fluctuations in tension have passed. The essential observation is the *minimum diastolic level*. This is usually observed twenty to forty seconds after the inhalation and characteristically just prior to the intense facial flush. A secondary rise in tension to levels equal or slightly higher than

management Failure to obtain results from treatment of early, spastic cases of hypertension clearly implies continuation of some provoking irritation

Predisposing etiologic factors, being largely inherent in the *patient's constitution*, are rarely amenable to therapeutic attack Early recognition of potential hypertension, often detected by the cold pressor test,²³ warrants prophylactic advice toward avoiding the more common sources of vascular insult The advisability of future pregnancies for such women as reveal signs of potential hypertension needs serious attention¹² Choice of occupation, relaxing utilization of leisure, avoiding dietary irritants, and encouragement in development of calmer emotional characteristics are questions the wise physician will bring up early in treating those individuals potentially hypertensive This is part of preventive geriatrics

Provoking etiologic factors, such as foci of infection, emotional turbulence, intoxication from metals, anemia, endocrine dysfunctions, including climacteric involution, excessive fatigue, and the like, are more readily affected by treatment Frequently *psychosomatic factors* are most significant and the physician can accomplish much by tactful but thorough exploration of mental conflicts⁸ Formal psychoanalysis is rarely, if ever, necessary²⁴ Continued worry and fatigue are probably the most common provoking factors, individual personality plays a most significant etiologic role Thus every patient must be handled differently Frequently, mild sedation over a long time is conspicuously beneficial Details of therapy directed against the more obvious organic etiologic factors are unnecessary here

It is possible, however, to lay down some general rules of hygiene and diet Moderation and temperance are the basic rules Insistence on radical and abrupt alterations in living habits is most unwise, particularly with older people Much mischief may be done by the abrupt deletion of coffee or tobacco, for their use is usually a deeply ingrained habit Coffee and other stimulants are contraindicated late in the day, for they are prone to interfere with proper rest at night In the morning, however, the mental stimulation and the diuretic and cardiac effects of caffeine are desirable The probable acquisition of considerable tolerance in older persons must not be forgotten

Tobacco, likewise, is best employed in moderation and the degree of curtailment must be adjusted to the fixity of the habit, the evidence that tobacco smoking actually affects the cardiac rate and rhythm, and the extent of habituation The enjoyment of smoking is a source of desirable mental relaxation It is doubtful whether the use of tobacco is significantly deleterious in hypertensive disease The majority of those who smoke are intense, driving personalities and any relationship between cause and effect must take this element into consideration

Alcohol, moderately enjoyed, is distinctly beneficial (see p 455) By inducing mental relaxation and encouraging peripheral vasodilation it has direct

valuable relaxing energy of alcohol is not necessary experience that

limiting themselves to a preprandial cocktail or brandy and a bedtime toddy adds to their well being

The *diet* in hypertensive disease has long been a matter of controversy Fads and theories have swung from one extreme to the other, but present con-

sensus is agreed on a sane middle course *moderation*. It should be remembered that hypertensive disease is a chronic disorder, requiring continuous management for years, and that therefore radical dietary restrictions are as unwise as are unlimited excesses. *The diet must be ample to maintain normal nutrition* and sufficiently palatable to be followed. There is no justification in curtailing protein intake below the normal metabolic requirements, approximately 1 gm protein per kilogram body weight. Undue protein restriction, especially if prolonged, depletes the body reserves and contributes to an exaggeration of any coexistent anemia. Albuminuria is *not* an indication for protein restriction, rather, because of the constant loss, the protein intake should be increased. It appears immaterial what the sources of protein are, meat, eggs, milk and cheese may be freely allowed. The evidence that cholesterol in eggs fosters arteriosclerosis in man is still controversial. Many elderly hypertensives are found to be inadequately nourished. There happens to be a patient attached to the disease. Treatment must consider the patient as a whole and not merely the disease.

Condiments and spices are frankly unnecessary dietary constituents. They contribute neither calories, minerals nor vitamins. Meat extractives (broths, bouillons, gravies, and consommés) are likewise almost nil in food value and are best deleted or reduced to the minimum compatible with a palatable diet. These items are potential renal and vascular irritants. Careful inquiry will reveal that a very high percentage of hypertensive patients habitually use condiments to excess.

Salt, on the other hand, is a necessary dietary adjunct. For a time a salt-free regime was advocated with great zeal, but there is much evidence indicating that the normal use of salt has no appreciable influence upon the course of the disease. Specific advice to the effect that the salt ordinarily used in cooking

consumption of water is very common among hypertensive patients in general and among the elderly in particular. Water is probably the best, and certainly the safest, diuretic. It is sadly curious how commonly the question of fluid intake is ignored in presenting dietary and hygienic advice. In instances of cardiac embarrassment (asthenic stage of hypertensive heart disease) water should be taken in small quantities at frequent intervals. While the heart is sthenic an average daily fluid intake of 2.5 to 3 liters is desirable. It matters little how the water is camouflaged, weak tea, fruit juices, milk, soups (preferably vegetable), carbonated beverages, and the like may be suggested for those objecting to drinking unadorned water.

The obese hypertensive should reduce. Obesity is decidedly antagonistic to longevity. Weight reduction is often accompanied by a fall of the arterial tension. Obesity greatly increases the work of the already overtaxed heart. It is always possible to accomplish reduction of weight by dietary measures. The intake of fats and carbohydrates must be diminished until the caloric value of the diet is just a little below requirements. Protein starvation is to be avoided. Gradual reduction is far better than attempts at quick slimming for several reasons.²⁵ In the first place, weakness and the distress of real hunger are avoided, secondly, weight lost slowly is much less likely to be regained and, thirdly, the skin and tissue turgor is less affected. The body, apparently, be-

comes adjusted to gradual consumption of the surplus of stored fat and the excessive appetite wanes. It is wise to limit reduction to a maximum of two pounds per week, one pound is better. In this manner a year's regime may accomplish a weight reduction of fifty or more pounds without distress or hazard. Weight reduction programs must be individualized and specific. Stout Mrs. Jones may complain that she eats no more than slender Mrs. Smith. Perhaps this is true, perhaps not. At any rate, it must be pointed out to obese Mrs. Jones that she *must* be eating more than *her* body requires, irrespective of her friend's diet. She may be spending less energy, assimilating her food more effectively, or retaining more water in her tissues than her envied neighbor. Nevertheless, she has been eating too much. If she consumes but 95 per cent of the calories used per day, the remaining 5 per cent must come from her store of fat. Trial and error and infinite patience may be necessary before the optimal reducing diet is fitted to the individual patient. Bread and pastries are the common vices of the obese.

Reducing the Burden of Injured Structures. Rest. Rest is the most ancient treatment. To apply it is instinctive and thus its origin antedates logic. Rest has both qualitative and quantitative attributes. Rest may be localized as in the immobilization of fractured bones or generalized as in sleep. Mental rest is sometimes best obtained through vigorous physical activity. The value of rest depends not only on the *location* and the *degree* of rest, but also upon its *duration*. Complete rest of the circulatory apparatus is impossible. The fatigue of the spastic arteriolar medial musculature and of the myocardium are both lessened by arteriolar relaxation, however. The primary concern is with the average arteriolar tension rather than with transient elevations. Reduction of the diastolic pressure is the guide to the extent and duration of vascular relaxation. There are four different approaches: (1) psychotherapeutics, (2) medicinal therapy, (3) physical therapy, and (4) surgical measures. There is no reason to limit treatment to any one category.

No single or specific "remedy" exists. Nor will one be found, the etiology of hypertensive disease is too complex. The management of each patient is an individual problem. Before advising any therapeutic measure, no matter how apparently trivial or how serious, one must weigh the probable benefits against the probable detriments. If the two are about equal, have the courage not to interfere.

Psychotherapy. In chronic disorders where management to control and/or retard the disease is a matter of years, it is most important that a wholehearted cooperation between patient and physician be established. It is up to the physician to understand the patient fully. To gain and retain the patient's confidence is half the battle won. One must frequently combat a deep resentment

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of creative hobbies, healthy and productive investment of leisure, and im-

indications and limitations of various medicinal agents vary in individual case problems

Sedatives are often useful in reducing the tendency to fretting and restlessness. Small doses should be prescribed over a long period of time. The duration of rest is most important. Frequently patients discover that their mental efficiency is increased by mild sedation. The bromides in small doses are useful. The barbiturates are less desirable for long-continued sedation, they are poorly tolerated by elderly persons. Alcohol is an excellent sedative if judiciously consumed. Recent, as yet unpublished, studies have shown that dilantin sodium in doses of $\frac{1}{2}$ grain (30 mg) three times a day may be most helpful in calming without depressing anxious patients.

Vasodilator drugs are on the most part too transient and fleeting in their effects to be of much value for long-continued administration in hypertensive disease. The alkyl nitrates, sodium nitrite, and amyl nitrite are invaluable in acute vasospastic emergencies, such as angina pectoris or cerebral spasm. Erythrol tetranitrate and mannitol hexanitrate have more prolonged action.

long enough to accomplish any lasting benefit.

More prolonged, gentler vascular relaxation is obtained from bismuth subnitrate,^{6, 9, 10} sodium or potassium thiocyanate, and the xanthine derivatives. The action of the latter group, particularly theophylline ethylenediamine (aminophylline), however, is distinctly selective, being limited almost solely to the coronary vessels (see p 417). Thiocyanates do induce the prolonged arteriolar relaxation of the type most desired in chronic hypertension. Therapeutically effective doses are precariously toxic, however. The margin between benefit and detriment is so close that thiocyanate medication is not justified unless the patient is under exceptionally close observation. Frequent deter-

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Bismuth subnitrate is nontoxic, noncumulative, inexpensive and easily

subnitrate, of the same order as occurs with sodium nitrite, has been demonstrated.²⁹ It is best prescribed in capsules of 0.6 gm (10 grains) each, taken three times a day. Bismuth subnitrate is of value as a mild vascular sedative in early (spastic) hypertensive disease. This adjunct action, however, is too mild to be of any great value in instances where provoking factors are actively operating.

Many other drugs have been tried. Calcium and cobalt salts were once in vogue. Iodides are probably useless. Parenteral magnesium sulfate reduces both the arterial and intracranial pressures. It is thus useful in some of the

acute complications of hypertension, such as cerebral edema, preeclampsia, and retinal exudates. The elimination of magnesium salts is much delayed when renal impairment exists, caution in this connection is necessary with older patients.

Physical Therapy. Many of the measures of physical therapy promote general relaxation and are thus most useful auxiliaries in the treatment of hypertension. Warm baths, sunshine, and massage are particularly useful, the reduction in blood pressure following diathermy is of short duration. Sudden changes of external temperatures are dangerous, especially with older and arteriosclerotic patients. A cold plunge may induce precarious vascular constriction. Massage, especially of the lower extremities, is of value in promoting relaxation.

When the expense involved is a cause for worry, more harm than good results. The major elements of spa treatments are relaxing baths, leisurely and pleasant walks.

is always available.

Surgical Measures. In the last decade, several neurosurgical procedures for the treatment of hypertension have been advocated, studied and revised. Extensive thoracolumbar sympathectomy induces a decided fall in arterial tension through paralysis of splanchnic and visceral vasotonic nerves.³⁰ Of the several operations devised,³¹ that of Smithwick appears to be the most effective, though there is, as yet, no general agreement. Therapeutic results, if evaluated after several years after operation,³² are not so good as implied by earlier reports. The most logical method of evaluating therapeutic results is in percentage approach toward normal of the arterial tension, not in mm. Hg of reduction. The duration of follow-up is important.

tempting to compensate for the splanchnic vasomotor paralysis, may induce the phenomena of Raynaud's disease. Certainly conservative medical management, with exceptionally painstaking attention to the various factors and their conscientious correction, should be advocated.

neurosurgical intervention. Extensive disease is slow, particularly in the elderly, and there is no need for haste. In so-called malignant hypertension, evidencing rapid progression, especially in younger patients, extensive thoracolumbar sympathectomy deserves a place in our therapeutic procedures.³³ Surgery is also useful in hypertensive disease in eliminating chronic foci of infection, in the treatment of hyperthyroidism, removal of adrenal tumors and the like. About one case per hundred thousand of hypertension is due to an adrenal tumor. The enthusiasm for unilateral nephrectomy when there appears to be asymmetric renal disease (hydronephrosis, pyelonephrosis) has already begun to wane. The possible benefits do not warrant the obvious surgical risk, particularly in elderly patients. The older the patient, the more likely is the progression of the disorder to be slow.

Improvement of Tissue Nutrition and Respiration Recalling that *the damage done by hypertension arises through interference with the nutrition and respiration of parenchymatous cells*, serves to emphasize the importance of this form of therapeutic attack. If hypoxemia be superimposed upon the vascular obstruction, the respiratory deficiency of the tissues is tremendously increased. Anemia, cardiac incompetence, and pulmonary disease are the major organic menaces to proper oxygen transport. Effort, by increasing the physiologic demand for oxygen, or residence in high altitudes further aggravates the tissue ischemia.

The management of cardiac decompensation (p. 387), chronic pulmonary disease (p. 316), and acute diseases of the lungs (p. 344) is discussed elsewhere. Anemia is frequently observed in hypertensive individuals. The coexistence of such anemia increases the tissue injury seriously, it is not necessary that the reduction in hemoglobin content be great in order to be of considerable moment. Correction of anemia, even if but of minor grade, is often of the greatest therapeutic benefit in hypertension, as in nephritis (p. 596). The hemoglobin should be rechecked periodically, for in many patients it will insidiously slip down again when antianemia therapy is discontinued (p. 203). There are many instances in which the arterial tension falls concurrently with a rise in hemoglobin values, in the absence of any other form of treatment. In older patients minor degrees of anemia and the subsequent hypoxemia are of much greater significance than in younger persons. It appears that aging (senescence) is associated with a diminishing tolerance to oxygen lack. The therapy of anemia is discussed elsewhere (see p. 196).

The nutritional requirements of the patient demand close attention (p. 187 and p. 189). As hypertension requires management for years, the basic diet must be adequate. Protein restriction must never be below requirements, protein starvation is often a factor in the anemia observed in these patients. The vitamins are important (see p. 190). A relatively high blood sugar is safer than lower levels in senile diabetics (see p. 221). Glucose is the major fuel for the heavily taxed cardiac muscle.⁶ Recognition of the importance of these many elements and the conscientious insistence upon a diet as nearly optimal as possible adds greatly to the efficacy of management.

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Success in the treatment of hypertensive disease is dependent upon meticulous attention to *many* details. A high degree of individualization in management is essential. The more thorough the search for contributory etiologic factors the more specific can be the treatment. All therapy must include the three fundamental forms of attack: (1) amelioration of causes, (2) rest for the

environmental factors must not be forgotten. It is the whole complex personality of the patient, not the disease alone, which must concern us.

HYPOTENSION

tension" applies to blood pressures distinctly below previous levels, but not necessarily below the normal, it implies previous hypertension. The three major factors responsible for abnormally low arterial tension are (1) depression of vascular tone, (2) diminution of cardiac force, and (3) loss of blood volume. These pathogenic changes may occur singly or, more frequently, in combination. Many provoking etiologic factors may induce hypotension. It is a frequent phenomenon in many diseases.

Hypotension may be acute, subacute, or chronic. The causes may be quite obvious, as in severe traumatic shock with hemorrhage, or obscure as in so-called "primary" or "essential" hypotension.

Types of Hypotension. Acute hypotension is conspicuous in coronary occlusion with left ventricular infarction (see p. 412), many types of poisoning, hemorrhage, shock, and following overdosage with vasodilating drugs. Subacute hypotension (lasting several days or weeks) is part of the clinical picture of such disorders as cardiac decompensation, acidosis, acute infections, anemia, dehydration, heat stroke, and intestinal disease. Chronic hypotension is a characteristic phenomenon in cachectic states (tuberculosis, anorexia nervosa, the inanition of cancer), Addison's disease, pernicious anemia, and many other debilitating disorders. The so-called "primary" chronic hypotension is associated with an asthenic, hypoplastic physique, small "drop heart," and fatigue. There are also many other, rarer, medical problems in which hypotension may be a factor.

Acute Hypotension. Acute hypotension is a major phenomenon of shock. The hazards, causes, and treatment of shock are not greatly affected by aging except that the relatively inelastic arteries of senescent persons make loss of circulating blood volume an even more serious menace than is the case when the arterioles can constrict quickly. Any acute and sudden fall in blood pressure in an elderly patient is potentially dangerous, whether the fall be to absolute low levels or merely low relative to the previous tension. Thrombosis in tortuous, roughened sclerotic vessels precipitated by acute slowing of the blood flow is a major risk.²² For this reason the choice of anesthesia (see p. 157) and the pre- and postoperative care of older subjects (see p. 148) are particularly significant in surgery.⁹ Spinal anesthesia is especially hazardous be-

of shock, however.

Subacute Hypotension. The subacute phenomenon is not the major hazard of shock, however. It may be a result of the shock itself, or it may be a phenomenon of its own. It is characterized by a gradual fall in blood pressure, often to levels below 90 mm. Hg. The patient is usually in a state of prostration, apocamnosis, profuse perspiration, cold, clammy hands, sense of

impending syncope (or actual attacks of syncope), and anorexia may last for weeks after an apparently mild bout of influenza. It is characteristic of this disease that the "hangover" is all out of proportion to the apparent severity of the attack. This is often increased by inadequate time for convalescence. With aging comes a slowing of recuperative powers. Diet, correction of anemia, liberal fluids, and small doses of strychnine will accelerate recuperation. Time, however, has no substitute.

Chronic or Habitual Hypotension. This is not uncommon. The so called "primary" and "secondary" forms differ only in the obviousness of their etiology. No disorder occurs without reason. Because the factors responsible are obscure is no excuse for failure to make an effort to search them out. Hypotension consequent to tuberculosis, the cachexia of cancer, or starvation is of little moment, the primary disease will destroy the patient first. Habitual hypotension unassociated with such obvious diseases is usually observed in those of hypoplastic physique, and is very frequently associated with chronic fatigue. Frequently these patients exhibit evidences of mild hypothyroidism, with basal metabolic rates in the neighborhood of minus 20. This is not surprising, for the chief clinical phenomenon of hypotension is a lowered endurance. Vertigo, chilly extremities, anorexia, and occasional syncope may be annoying but the major complaint is a constant sense of fatigue. The prognosis for longevity is excellent, the outlook for full energetic vigor is dubious when constitutional factors predominate in the etiologic picture. The life expectancy of people with uncomplicated constitutional hypotension is better than the average according to life insurance data.

Treatment of Hypotension. The treatment of such chronic hypotension includes the three fundamental principles discussed in connection with hypertension: etiologic therapy, rest, and measures to insure as optimum a nutritional status as possible. Little, if anything, can be done about the inherent constitutional factors which are etiologically responsible. It is of paramount importance that these people learn their physical limitations and avoid pushing fatigue over into exhaustion. Short rest periods, occupations suited to individual capacity, recognition that an excess of ambition is futile, for it leads but to exhaustion, and encouragement toward outdoor physical rather than mental activity will all contribute to a greater sense of well being. The lack of arteriolar tone is amenable to medicinal stimulation. *Epinephrine* is, of course, invaluable in acute emergencies of sudden and severe circulatory collapse, but it is impractical in the treatment of chronic hypotension, it must be administered parenterally and its action is far too transient to be of lasting benefit.

as read—

Although the basal metabolic rates of these patients are rarely conspicuously below normal, they tolerate small doses of thyroid gland substance well and are often made much more vigorous thereby, especially when the appetite is improved. Thyroid medication is never justified, however, without control observations of the basal metabolic rate. As a general tonic, stimulating the

appetite and enhancing muscular tone and vigor, *strychnine* is still the drug of choice. Elderly patients tolerate it well. Strychnine sulfate, 0.006 gm ($\frac{1}{160}$ grain) thrice daily, before meals may be continued for weeks with distinct profit. The old-fashioned elixir of iron, quinine, and strychnine is excellent, particularly when the patient is conspicuously undernourished. Many aged patients are aided by the continuous administration of small doses of digitalis.

Gain in weight is usually associated with improved vigor. When an atonic intestinal tract rebels at large meals, smaller but more frequent feedings are advisable. Sweets should be avoided, they tend to depress appetite. A relatively high protein diet and a liberal intake of all the vitamins is indicated. Particularly valuable are the newer capsules of concentrated vitamin B complex. Coincident anemia markedly exacerbates the symptoms of chronic hypotension. Anemia of moderate degree is extremely frequent in these people. Its correction may relieve most, if not all, the complaints despite a negligible change in the objective observations of the arterial tension.

Apocamnosis encourages sedentary habits. These people are often accused of laziness whereas in fact they feel truly fatigued. As age advances there are more and more excuses for inactivity. Yet outdoor exercise and sunshine do much to improve vigor, their sane enjoyment must be encouraged. Exercise must be graded to the individual patient's capacity, competitive sports are best avoided. Walking, fishing, horseback riding, and golf are excellent forms of exercise. The program should be continuous, sporadic sprees of exercise interspaced by long periods of extreme sedentary living do more harm than good.³⁴ Frequently there is greater hazard in underdoing than in overdoing for elderly patients.

SUMMARY

The problems of abnormalities of the arterial tension are among the most pressing questions of geriatric medicine. They involve, however, the aging far more frequently than the truly aged. The earlier in the course of these diseases control measures are instituted, the more effective is the therapy. More can be accomplished for the aging than for the aged.³⁵ The critical years are the two decades from forty to sixty years, if we can retard and control the diseases of senescence during these years, the health of the aged will be vastly improved and disability may be postponed until senility alone brings about infirmity. Such are the objectives of *preventive geriatrics*.⁴ Constant cognizance of the importance of health maintenance, rather than concern only with repairing the ravages of disease so often avoidable, must inevitably reduce the toll of disability and permit many of the increasing millions of the elderly to remain useful, and therefore happier, further into the twilight of senectitude.³⁶

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CHAPTER 31

DISEASES OF THE VEINS

ROBERT R. LINTON

VARICOSE VEINS

VARICOSE veins of the lower extremity are among the most common of the diseases affecting man. The etiologic factors producing them are a defect in the valvular mechanism of the leg veins and the increased pressure to which they are exposed due to man's erect position. Untreated varicose veins produce typical pathologic changes in the skin and subcutaneous tissues of the lower leg. These changes become more marked with increasing age, since varicose veins and their complications represent a progressive disease. First the veins become dilated and tortuous. The walls become greatly thickened. There is interference with the normal nutrition of the adjacent tissues, presumably because the blood in them stagnates. This results in fibrosis of the subcutaneous tissues. Small petechial hemorrhages may be seen in the skin of the lower leg. The exact cause of these is not fully understood, but presumably they are due to the increased pressure on the capillaries with resulting rupture. If the veins are untreated these hemorrhages continue to take place so that after a number of years the lower leg, especially on the medial side, takes on a characteristic brownish pigmentation. This condition is especially common in the elderly patient who has never received adequate treatment. Once the pigment is present it never entirely disappears even though the veins are eradicated.

The nutrition of the skin of the lower leg in an individual with long standing varicose veins may be interfered with to such a degree that if it is broken by an abrasion or a laceration there is great difficulty in healing the wound. This results in the so-called varicose ulcer which is one of the common complications of varicose veins.

Symptoms The most common symptoms of varicose veins are a sense of fullness and a tired feeling in the leg. A few patients complain of pain but this is not the most common symptom. Young persons dislike the appearance of the veins, whereas elderly patients care less about the cosmetic aspect so long as they are free of the sequelae, such as dermatitis, ulceration, and thrombosis. It should be remembered also that when an individual changes from a horizontal to an erect position pooling of large quantities of blood in the varicosities may occur, causing tachycardia and a sensation of faintness due to a reduction in effective blood volume. This is especially true in the aged who have a vascular system which compensates slowly to sudden shifts in the circulating blood volume.

Diagnosis The diagnosis of varicose veins is usually very obvious. The most useful test to determine which system or systems of veins are at fault, in order that the proper treatment may be carried out, is the Trendelenburg test.

In dealing with elderly patients there is one very important differential

diagnosis to make. This is the dilatation of the superficial veins of the lower leg or extremity which so commonly accompanies obliterative arterial disease due to arteriosclerosis. Careful examination of the extremity in this condition reveals that these dilated veins are much different than the usual varicose veins. As a rule they are not tortuous or thick walled (Figs 91 and 92). There is seldom any pigmentation of the skin. A careful history from the patient will usually elicit a story that they have appeared recently which would be unusual

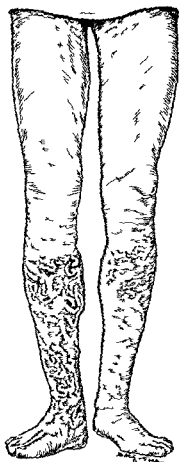


Fig. 91. Lateral varicose veins in a man aged sixty. Note that the saphenous trunk can be seen in the upper third of the thigh. The skin of the lower leg shows marked changes due to the long standing varicosities.

if they were simple varicosities. The skin of the foot and lower leg will present a deep reddish color with the late stages.

In such cases the therapy should be for the arterial disease rather than for the dilated veins.

Treatment. The treatment of simple varicose veins in the elderly patient should be approached with a somewhat different attitude than in a young or middle-aged individual. In general this group of patients seek treatment not because of the veins but rather for the sequelae. The use of the time-honored elastic stocking actually has a definite place in the treatment of varicose veins in the aged. It will give symptomatic relief by preventing the pooling of blood and also will control edema which frequently is found in the lower leg of an inactive elderly individual.

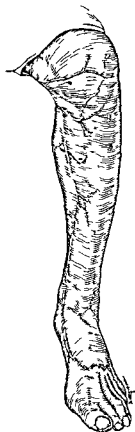


Fig. 92 Enlarged varicose veins in a man of sixty-eight with arterial insufficiency due to arteriosclerosis. This type of venous enlargement should not be confused with varicose veins due to incompetence of the venous valves. Note that these veins are not tortuous; they are simply dilated. Compare this figure with Fig. 91.

The most satisfactory treatment in an active, otherwise healthy individual up to sixty-five or seventy years of age is (1) interruption of the long saphenous vein at the saphenofemoral junction, (2) if the short saphenous vein is involved, interruption of it at the popliteal-saphenous junction, and removal of either or both of the main trunks of the long and short saphenous veins down to the ankle by a subcutaneous stripping procedure. Retrograde injections should not be performed because of the danger of producing a deep thrombosis and pulmonary embolism. Injections of sclerosing solutions before or after operation must be done with great caution because of this same danger.

If injections are to be used, it is advisable to utilize small amounts of the sclerosing solution. Any of the standard solutions are acceptable. Sodium morrhuate or sotradecol has the advantage of not producing sloughs so frequently as some of the others.

SEQUELAE OF VARICOSE VEINS

Rupture of a Varix. Serious hemorrhage due to the rupture of a varicosity may occur but it is rare. The treatment is simple. The patient should lie down with the affected leg elevated well above the level of the heart, in which position the bleeding will soon cease. A small gauze pad over the bleeding point held firmly with adhesive tape will control further bleeding. A more permanent result can be obtained by injecting the affected vein with a sclerosing solution a short distance on both sides of the bleeding point.

Varicose Dermatitis (Eczema) Varicose eczema is the name commonly given to an irritation of the skin that overlies varicose veins or surrounds a varicose ulcer. The skin involved is usually scaly and roughened, often showing excoriations where the patient has scratched it. Eradication of the varicose veins usually alleviates the condition.

Varicose Ulcer. An ulcer on the medial side of the lower leg just above the medial malleolus is one of the common sequelae of long-standing untreated varicose veins. The direct cause of the ulcer is usually trauma which produces a break in the skin, and because of the interference with nutrition, due to the varicosities, the lesion does not heal and eventually becomes a chronic ulcer. There are two types of varicose ulcers: (1) The simple ulcer associated with varicosities only of the superficial system. (2) The post-thrombotic varicose ulcer that appears in a leg which has previously been affected by thrombosis of the deep veins. This latter type will be taken up under a separate heading.

The basis of treatment of simple varicose ulcer is eradication of the varicose veins. At times this may be done as an ambulatory procedure, providing the ulceration is not too large. Frequently one may find a varicose vein feeding directly into the ulcer. An injection of a sclerosing solution into this vein close to the ulcer area will often produce a miraculous cure. At other times, especially in the aged, it may be necessary to resort to some form of restraining bandage, such as an elastic adhesive, or a gelatin paste bandage such as an Unna's paste boot. Another method is to place a rubber sponge over the ulcer area, first protecting it with a layer of gauze and then wrapping the leg and the rubber sponge securely with an elastic cloth bandage. In cases of long standing chronic ulcers it may be necessary to put the patient to bed and elevate the leg applying warm boric acid compresses. If the ulcer is very large a split skin graft may be applied. After the ulcer is healed appropriate measures, such as ligation and stripping of the saphenous vein, may be taken in order to obtain a more permanent result if the patient is not too aged, otherwise an elastic bandage is the best form of therapy.

Superficial Venous Thrombosis (Phlebitis). Intravascular thrombosis in a varicose vein is not an uncommon complication. It is more frequently seen in elderly patients because their veins become sclerotic and, as they are less active, the blood in the varicosities becomes stagnant and clotting more readily takes place. The exact cause which precipitates the thrombosis is not understood but not infrequently it follows trauma. It occurs most commonly in the dilated veins of the lower leg but it may appear first in the long saphenous

trunk of the thigh. The symptoms are pain and tenderness along the course of the involved vein with some swelling adjacent to it and redness of the skin over it. There is never generalized edema of the whole extremity. Careful palpation reveals a cordlike structure where the soft compressible veins should be. Superficial venous thrombosis may prove to be a serious condition if the clot in the saphenous vein propagates through the saphenofemoral junction or a



Fig. 93 Chronic ulceration of the lower leg in a woman aged sixty-seven of twenty-five years' duration. Note the skin changes about the ulcer and the narrowing of the leg in the lower third just above the malleoli. Treatment in this case was a low thigh amputation because of the marked changes in the skin and subcutaneous tissues of the lower leg and dorsum of the foot. The hypertrophied nail due to onychomycosis should be noted on the great toe.

communicating vein into the deep veins to form additional thrombi. This latter part of the clot may become dislodged to produce pulmonary embolism, which is a common complication of the disease. Fatal pulmonary embolism has been reported in the case of a patient with chronic venous disease. The following cases illustrate the complications of the disease.

The best *treatment* of superficial venous thrombosis regardless of the age or the level to which the thrombosis may have ascended, and even though a hard firm cord can be felt at the upper end of the saphenous vein, is an immediate ligation and division under local procaine anesthesia of the long saphenous vein at the saphenofemoral junction, or if the short saphenous vein is involved at its junction with the popliteal vein. If it is found that the thrombosis has extended into the deep system, the thrombus should be extracted by suction after making an opening in the deep vein. The superficial femoral vein

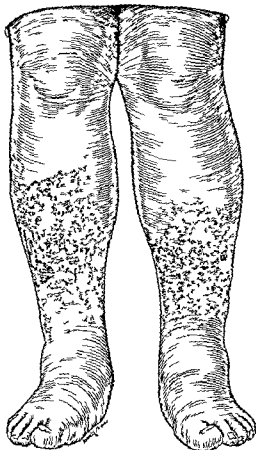


Fig. 94 Chronic lymphedema, dermatophytosis and ulceration following bilateral deep venous thrombosis in an elderly patient who has received inadequate treatment.

should be interrupted as well as the saphenous under these conditions. This operative treatment has the advantage not only of preventing pulmonary emboli but also of allowing ambulation immediately after the operation. The use of ice bags in the treatment of phlebitis is not recommended except for a patient's comfort; moist heat in the form of hot packs is preferable. These measures are usually unnecessary with the operative treatment as described.

The *prophylaxis* of superficial thrombosis is important. If an elderly patient with large varicose veins is stricken ill and must be put to bed or has a

major operation requiring bed rest for a prolonged period, the elevation of the extremities with the emptying of the superficial veins of the legs at two- or three-hour intervals will in most cases prevent phlebitis. Active exercises of the legs should be encouraged as soon as possible.

VENOUS THROMBOSIS OF THE DEEP VEINS

(Thrombophlebitis or Phlebothrombosis)

Venous thrombosis in the deep veins of the lower extremity occurs most often concurrently with an illness such as pneumonia or after some major surgical operation but it may be spontaneous. It is a serious threat to a patient's life since the thrombus may be dislodged to produce fatal pulmonary embolism.

Etiology. The thrombosis usually develops first in the veins of the calf. The exact etiology is not understood except that it is more apt to occur while the patient is in bed when the rate of blood flow in an extremity is relatively slow. The left leg is more commonly affected. Degeneration of the venous intima with age, similar to that seen in the arteries, may be an important etiologic factor in deep venous thrombosis. Early in the course of the disease there is little if any inflammatory reaction, so that it may be difficult to detect by a clinical examination. In this stage it is frequently called phlebothrombosis. Later as inflammatory signs develop it is called thrombophlebitis. Pain or swelling in the calf is the most common early clinical sign. The diagnosis frequently may not be suspected until the patient has had a pulmonary infarct or embolism. If the leg becomes rapidly swollen and painful the diagnosis is readily made.

The occurrence of a nonfatal pulmonary infarct demands a very careful search for deep thrombosis of the leg veins in order that measures may be taken to prevent further emboli. A patient with a greatly swollen leg from thrombophlebitis runs less chance of having pulmonary embolism than the patient who has phlebothrombosis without the swelling. This is because in the former the thrombus is usually adherent to the intima of the vein and as a result it is not apt to be dislodged, whereas in the latter condition the thrombus is not firmly attached so that for the most part it lies floating in the venous blood stream. It is this floating section which may break off at any moment to produce a fatal embolus.

The *prophylaxis* of deep thrombosis requires special emphasis since by it fatal embolism may be prevented. A fundamental point in this regard is to keep constantly in mind the fact that it may occur to any individual and that there is no way beforehand to detect in whom it may develop. Of special significance is the fact that the circulation in the extremities is most sluggish while a patient is lying in bed at rest. For these reasons it is important that any patient who is put to bed should be encouraged to exercise the legs as much as possible. If the patient is too sick to do this or if he has recently had a major operation, then the venous circulation must be stimulated by having the extremities raised periodically above the level of the heart so that the venous blood will be drained out of the legs and not remain stagnant in the veins.

Active exercises of the calf muscles seem to be the best preventative and they should be encouraged in bed patients. If possible following surgical operation early ambulation within twenty-four to forty-eight hours is advised.

The anticoagulants, heparin and dicumarol have a definite place in the prophylaxis of venous thrombosis, but they should be used with the utmost caution in the aged because of the danger of cerebral hemorrhage.

Treatment. The safest method of treatment of early deep thrombosis without pulmonary embolism in competent hands is bilateral interruption of the superficial femoral veins at the groin, performed under local procaine in filtration anesthesia. Heparin and dicumarol are advocated by some, but it is believed they are not so safe or effective, unless a competent surgeon is available.

The repeated injection of the lumbar sympathetic ganglia on the affected side with a 1 per cent solution of procaine is efficacious in some cases for the relief of pain and edema, especially in the large swollen legs. This therapy, however, will not prevent pulmonary embolism. The use of ice packs in the treatment of deep venous thrombosis should be abolished as this method serves no good purpose, since cold tends to increase the vasoconstriction. The use of heat is a well recognized form of treatment. It is best applied in the form of hot packs to the entire leg. These should be changed at two- to three hour intervals leaving them on for thirty minutes. The effect of this treatment is essentially the same as that of paravertebral novocain injection as it tends to relieve vasospasm.

In a patient who has had a pulmonary infarct with or without a recognizable venous thrombosis of the lower extremity there is only one treatment which should be considered, namely, the femoral vein in the groin should be interrupted to prevent further pulmonary emboli. If the thrombus is found to extend above the inguinal ligament, it should be removed by means of suction after opening the vein. Interruption of the femoral vein distal to the profunda femoris branch should produce less edema and disturbance of the venous circulation in the extremity, but from actual experience it seems to make little difference whether it is ligated here or above the profunda branch. The important point is that the common femoral and the long saphenous veins should not both be interrupted, especially in the aged since these patients have such a poor venous collateral circulation. Bilateral interruption should be done in elderly patients even though signs of thrombosis are only recognized in one extremity. The chief advantages of femoral vein interruption are that it prevents massive pulmonary embolism, and that the patient is ambulatory in most cases within twenty-four to forty eight hours after the procedure. Thus the patient's life may be saved and the convalescence from deep venous thrombosis, which ordinarily requires several weeks in bed, may be shortened to a matter of days.

The *after care* of the phlebotic extremity is important, whichever of the above forms of treatment has been instituted, since there is always a variable amount of edema when the patient is up and about. A well fitting elastic stocking from the toes to above the knee, or an elastic bandage up to the knee, should be worn during the day in order to reduce the swelling to a minimum. The elastic support should be worn until the swelling disappears. If there is any dermatophytosis on the feet or toes it should be eradicated by the use of fungicides. This is important because persistent edema plus fungous infection frequently leads to chronic lymphedema and ulceration of the lower leg (Fig 94).

PULMONARY EMBOLISM

Pulmonary embolism is an extremely serious condition especially in the aged patient. If the embolus proves to be a very large one, death usually ensues within a few minutes. The symptoms associated with this condition are a severe agonizing pain in the chest, often behind the sternum. The patient suddenly goes into collapse presenting the classical signs of shock. In many cases it is very difficult to differentiate between pulmonary embolism and coronary infarction. Even with the aid of the electrocardiogram it is sometimes impossible to differentiate the two conditions. If the patient survives, the raising of bloody sputum is practically pathognomonic of embolism. An x ray of the chest twelve to twenty four hours after the onset of pain gives very valuable information, as the picture may show a characteristic triangular area of infarction. The seriousness of the situation depends not only upon the size of



Fig 95 Anteroposterior and lateral x rays of chest in a man who suffered recurrent pulmonary infarcts more marked on the right side. A healed infarct will be noted on the left side as a horizontal line opposite the apex of the heart. This case was confused and was mistaken for a lung tumor for a long while. The patient was completely relieved of further infarcts by a femoral vein ligation and division with aspiration of the thrombus from it.

the embolus but the age of the patient and the condition of the heart. This is because the embolus tends to obstruct the outlet from the right heart, throwing a terrific burden upon it which it cannot stand. The result is death from heart failure. An embolus which would be sublethal in a young healthy individual might prove fatal in an older person.

Treatment The immediate treatment of pulmonary embolism if it does not prove fatal is sedation with morphine and placing the patient in an oxygen tent in order to prevent anoxia and the resulting effects upon heart and body in general. Venesection with the withdrawal of 500 cc. of blood has been reported to aid in relieving the right heart. The use of the various vasodilators such as papaverine has been recommended, but these are of secondary importance to the measures already mentioned. The treatment of massive pulmonary embolism by operative means, opening the pulmonary artery with removal of the

clot, is not recommended in elderly patients, as the heart in this group of patients will not withstand such an operation

Once the acute symptoms of a pulmonary embolus have been controlled in the nonlethal case, the most important thing to do is to ascertain the source of the embolus. This means a very careful investigation of the lower extremities. If a superficial venous thrombosis, involving the long saphenous vein, is discovered a ligation of the vein at its junction with the deep system should be made and in addition the superficial femoral vein of both extremities should be interrupted. If the thrombosis is in the deep veins the femoral vein should be ligated in the groin distal to the profunda femoris branch unless a clot extends above it. Under these conditions it is best to aspirate the thrombus and interrupt the femoral vein distal to the saphenous vein. These operative procedures are relatively simple and yet are most effective in preventing further pulmonary embolism. In rare instances of septic pulmonary emboli arising from a septic thrombophlebitis in the pelvis and with pulmonary embolism in the presence of phlegmasia alba dolens, interruption of the inferior vena cava is indicated. As this is a more shocking procedure it should be done only in such cases and only if the patient's condition is satisfactory.

CHRONIC ULCERATION OF THE LOWER LEG

(Post-thrombotic Varicose Ulcer)

Chronic ulceration of the lower leg is one of the distressing sequelae which develop following deep venous thrombosis. It is frequently seen in the elderly patient. The ulceration commonly occurs one to two years after the thrombosis, but it may not appear for twenty. The typical location of the ulcer is just above the medial malleolus on the inner side of the lower leg. It is characterized by its chronicity and the difficulty in obtaining a cure. There are many forms of temporary treatment but only radical surgical procedures will produce a cure in most cases. This condition usually begins as an indurated, painful area which on examination is found to involve chiefly the subcutaneous tissues. Microscopic examination of these tissues shows a nonspecific inflammatory reaction. Later, as the condition progresses there may be a brownish pigment deposited in the skin. The nutrition is finally interfered with so that a slight abrasion produces a chronic ulceration. The typical post-thrombotic varicose ulcer is exquisitely painful. There is a copious discharge of semipurulent fluid. The ulcer may vary from a few millimeters in diameter to one which involves the entire circumference of the lower leg extending from the malleoli to the mid lower leg. In coexistence with the ulceration there is usually a marked dermatitis. Examination of the feet of these individuals shows frequently a fungus infection of the toenails, and sometimes of the skin of the toes and feet that may play a role in the etiology of the chronic ulceration. The ulcer bed is so well protected with granulation tissue that seldom does an overwhelming infection enter the body through it. Bacteriologic study usually shows a mixed growth and in some cases even hemolytic streptococci.

Etiology. The etiology of this type of ulceration has not been completely established. A damaged venous system, the result of deep thrombosis, plays a very important part. By the time the ulceration has developed the deep veins of the leg in most instances have canalized. The valves have been destroyed by the thrombotic process so that the hemostatic pressure exerted on their walls is greatly increased. This increased pressure is transmitted to the communicat-

ing veins, which produces dilatation with resulting incompetence of their valves. Venous stasis occurs in the large veins with resulting interference to the nutrition of the skin and subcutaneous tissues. In this type of ulcer, therefore, we are dealing with systems of incompetent deep and communicating veins rather than just the superficial system as in simple varicose ulcers. The involved extremity in individuals with post thrombotic varicose ulcers presents a certain amount of edema which is an aftermath of the deep venous thrombosis and probably plays a part in the chronic ulceration. All patients who have had deep venous thrombosis do not develop the post thrombotic type of varicose ulceration. It is seen chiefly in the poorer classes and in those in whom bodily cleanliness is neglected. Malignant degeneration in varicose ulcers is a rare complication. In a series of three hundred cases recently studied at the Massachusetts General Hospital it occurred in but one patient.

Treatment. There are many forms of treatment for this type of ulceration. Prophylaxis is an important measure. If a patient has had a deep venous thrombosis he should be instructed to control the edema of the extremity by means of an elastic stocking. He should wear this for at least a year and some times longer if the edema persists. If it is allowed to persist it is practically impossible to cure it after several years and the chances of developing a chronic ulcer are much greater than if the edema is controlled. A careful search for fungus infection of the toes should be made and if present it should be eradicated by an appropriate fungicide.

When the ulceration is already present it is sometimes possible to control and heal it by means of various types of bandages and ointments. The most

terial circulation is adequate and his age is not too great, then operative measures can be instituted which will produce a cure if properly carried out. Excision of the ulcer with the subcutaneous tissues and deep fascia down to muscles produces many excellent cures. Another method is to interrupt the superficial femoral veins, ligate, and remove by stripping the long and short saphenous veins. The results of lumbar sympathectomy have been disappointing. This is because of the extensive secondary changes in the skin and subcutaneous tissues. Local skin grafts without excision of the ulcer are of no value because the new skin disappears rapidly when the patient becomes ambulatory.

SECTION VI

DISORDERS OF THE ALIMENTARY SYSTEM

CHAPTER 32

DISEASES OF THE MOUTH AND TEETH

JAMES BARRETT BROWN AND MINOT P. FRYER

TUMORS OF THE MOUTH

CARCINOMA is the most important, most dangerous, and most commonly considered disease in the mouths of older people. It is more curable in older than in younger patients. This point is sufficiently important to urge making every effort not to miss the diagnosis of buccal carcinoma. It is also significant in prognosis.

Pharynx and Base of Tongue. Carcinomata of the pharynx and the base of the tongue are frequently not diagnosed because these growths are apt to be silent as to pain or discomfort, or if symptoms do occur, they are apt to be associated with other diagnostic ideas than cancer. In routine physical examinations of older persons, these areas should always be observed carefully. Any slight pain or discomfort, or bleeding, should make one suspicious. If nothing can be seen, slip the finger over the areas and down the side of the tongue, using an application of local anesthetic if needed. The growth usually appears as an ulcer with some piled-up tissue with hard edges. Although it may be painless, examination with the finger usually produces discomfort. Biopsy should be done if there is any question about the lesion. Biopsy is rarely difficult with either a biting forceps or a diathermy loop after adequate local anesthesia.

These growths in the base of the tongue and pharynx are usually of high grade malignancy and metastasize early. Most commonly they form a mass under the angle of the jaw. Sometimes there is so much local growth that this mass is a direct extension rather than a metastasis. Such a development is, of course, a late and bad one.

or transitional cell carcinomata. This differentiation has little to do with diagnosis but does have something to do with prognosis and treatment. A low grade squamous cell carcinoma is, of course, more curable than a high grade one of poor differentiation, but these transitional cell carcinomata are radio-sensitive and often disappear completely and quickly under proper radiation treatment. They are, however, more apt to return than the better differ-

entiated types. Adenocarcinoma occurs occasionally. This group of cancers is not a very well recognized one and really does not need to be separated except for the original suggestion that they are probably the most commonly overlooked of all growths in the mouth.

Treatment is usually by interstitial radiation in the form of gold radon seeds, both for the original mass and the metastases. Neck dissection is done when there is a reasonably good chance of controlling the local growth, when the metastases can be removed, and when the age and physical condition warrant a radical procedure. Protracted external x-ray radiation may be still preferred by some, but it has so often been ineffective that prejudice has developed against it. This is not warranted, for good results can be obtained by properly instructed radiologists working with proper equipment.

Tongue and Floor of Mouth. Carcinomata of other areas of the tongue and of the floor of the mouth may be grouped together. They are prone to follow the same silent course as those arising from the base. The first thing the patient may notice is a mass in the neck which has been called to his attention by some friend or a barber. This may seem a trivial point, but it is not, if the valuable time that has been lost in diagnosis and treatment is considered.

Diagnosis. The lesion in either area is apt to be a fissured ulcer with extra tissue piled up along the edges. The ulcer margins may be overhanging, bleed readily, reveal sloughing and may or may not be painful. Cancer in the body of the tongue itself may be a solid type of growth, usually with ulceration. Less frequently it is superficial, with a fungating mass occupying the center.

The further forward in the tongue the growth arises, the better is the prognosis, as a rule. This dictum does not hold for cancers arising in the floor of the mouth. Adenocarcinoma is a little less rare in the body of the tongue than at the base. They may develop rather extensively without ulceration.

Treatment consists of interstitial radiation with gold radon seeds, plus early and complete dissection of the neck on the involved side. If metastases are already present and known to be fixed, and operation is generally contra-indicated, then radon seeds should be planted in the involved areas in the neck. The methods of radiation vary a great deal in different clinics. No qualitative or quantitative discussion can be entered into here, or need be.

The time at which the neck dissection is done is of importance. Because of the tendency to metastasize early, and because fatal metastases may arise following treatment of the original lesion while one is waiting to see if primary healing will occur, the surgical search for metastases in the neck is often best carried out while the radon is still inside the mouth, or at least within a month, whether primary healing has occurred or not.

Carcinomata of the Alveolus, Cheek, and Jaw. These are grouped together because of association in involvement. The jaw, of course, is involved only secondarily, but there is a tendency to use the term "carcinoma of the jaw" loosely as though the growth were primary in the bone. These areas are usually involved with growths of lower malignancy which are apt to follow some preexisting leukoplakia or irritation around the teeth. They are frequently wart like or verrucous, fungating outwardly, and are therefore more curable than other carcinomata arising inside the mouth. Upper alveolar tumors may slowly extend to the palate and be silent as far as symptoms are concerned, just as the first mentioned type of cancer. (See Figs 96 and 97.)

Removal by diathermy is often the best treatment of these lesions. If the

bone is involved seriously, or if there will be too much lining of the mouth lost to allow normal opening then the bone should also be burned and either re-



Fig 96 Carcinoma of the buccal mucosa with widespread involvement of the jaw bone which was mistaken for osteomyelitis

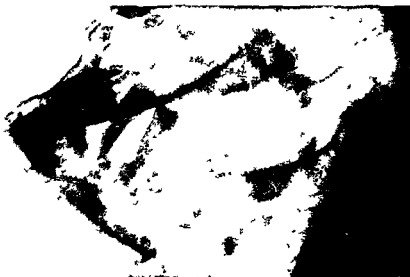


Fig 97 Fracture through early involvement of squamous cell carcinoma. The first symptom had been loose teeth, which were pulled and the jaw broken at the time. Six weeks longer were occupied in delay of treatment for supposedly infected fracture

sected at the time of the operation or allowed to sequestrate and be removed or thrown off later. When a wide involvement occurs and a large removal must

be done, an upper neck dissection is sometimes combined with it and the whole procedure is a modified Bloodgood operation. This usually requires a secondary oral fistula repair. The missing jaw can seldom be replaced, the opposite side must be depended upon for function.

In the upper alveolus the cancer growth may spread rapidly into the antrum and up into the zygomatic fossa. For this reason the antrum is often opened, inspected, and further diathermy removal carried out. The antrum should be left open into the mouth for several months so that the area may be reinspected. The defect can be covered with a dental plate or repaired with a palate flap when one feels sure there will not be a recurrence. As always, radiation therapy has to be considered, but it is generally best not to use interstitial radiation too close to bone because of the slowness of healing and pain. Ex-



Fig 98 A fungating carcinoma missed on the inside of mouth because patient had syphilis thought to be osteomyelitis when it appeared externally

ternal radiation or contact x-ray inside the mouth may be relied on if operation is contraindicated, or a combination of both may be used.

Carcinoma of the Lip Little discussion is needed here except to urge early and complete treatment. This is the easiest of all carcinomata to diagnose, although it does occur in different forms. There may be a fissure, a flat ulcer, or a chronic leukoplakia that finally becomes malignant, or a fungating mass of short duration. In *treatment* there is a marked tendency to rely on radiation alone. Surgical excision still has its place, especially as it affords opportunity for thorough microscopic studies. Instead of the usual wide V-excision, an

sive tumors the whole lip should be removed and a late repair made from the upper lip cheeks or from a distant flap (See Figs 99 to 102)

Small active carcinomata can be well treated without excision. The whole lip can be radiated with interstitial radon, surface radium, or contact x rays.

Neck dissections should be considered for all old lesions, for large ones and, of course, when metastases that are thought to be operable are present. Carcinoma of the lip is thought by many to be easily curable. It is, with early and proper therapy, but when once it has progressed the patient suffers the loss of an entire feature, and when metastases are allowed to develop, the threat of death is the same as with any other carcinoma.

Carcinoma of the upper lip may be of the basal cell variety. It usually is amenable to cure either with radiation or surgery. Here again, if the growth is inadequately treated or becomes invasive in character, the patient will have a most protracted illness. If the growth extends deeply in along the side of the



Fig 99



Fig 100

Fig. 99 Squamous cell carcinoma of the lip with complete healing following an oval cauterization, radium treatment and bilateral upper neck dissection. Patient well nine years.

Fig. 100 Same patient as in Fig. 99 after surgical excision healed.

nose it becomes increasingly resistant to treatment. This is the typical "rodent ulcer" of old people (See Figs 101 and 102).

Biopsy Study of Lesions of the Mouth If a growth or ulcer persists for two weeks (at the most for four weeks), is resistant to ordinary treatment, and its nature is not plainly carcinomatous, then carcinoma should be suspected and a biopsy done without further delay. If the lesion is small enough, the whole mass might as well be removed, rather than only a part. The diathermy loop is ideal for these biopsies, but if a very small piece is to be taken, it should be cut away sharply and the resultant base cauterized. This procedure permits one to obtain a specimen of tissue undamaged by heat. If only part of a growth is being excised for examination, it is usually well to include tissue from the edge of the lesion. It is further to be recognized that biopsies are not infallible and neither are pathologists' reports. Therefore, if a growth is clinically carcinoma, it should be treated as such regardless of a "negative" laboratory report.

One rather frequent source of error in the diagnosis of oral cancer is the *coincident occurrence of syphilis*. There is a strong inclination to assume that all buccal lesions are syphilitic when the disease is known to be present. As a result, much valuable time may be lost, and the treatment of the cancer delayed. Obviously cancer is more urgently important to the patient than syphilis.

Microscopic Grading of Carcinoma This is carried out routinely by nearly all pathologists in their description of malignant tissue. The four grades listed by Broders have been adopted in many laboratories. These grades describe the degree of malignancy of the cells on the basis of their lack of differentiation. Grade I is the least malignant, 75 per cent of the cells are sufficiently differentiated so that they can be recognized as squamous cells with intercellular bridges, pearly formation, good polarity and hornification of the individual cells. Grade II is 50 per cent undifferentiated. Grade III is 75 per cent,



Fig 101



Fig 102

Fig 101 Basal cell carcinoma of upper lip when first seen having been intractable to both surgery and surface radiation with complete retrogression of growth following heavy interstitial radon treatment. Patient well ten years.

Fig 102 Same patient as in Fig 101 after surgical excision.

and Grade IV shows from none to only 25 per cent of the cells differentiated. This is a purely personal quantitative finding and does not have the prognostic value that has been claimed for it. For one thing, in a large growth all four grades might be found in different areas; if one had enough biopsies. The final grade should be considered as the highest found, not as the lowest. The area involved has a great deal to do with the prognosis. *e.g.*, a Grade IV on the lip (which is unusual) might not be any worse than a Grade I on the tongue (which is also unusual). The size of the growth and its duration must also be considered in estimating the likelihood of metastasis, irrespective of whether the cancer is of low grade or not. For example, a carcinoma of the lip that has been present from six to nine months is much more apt to have metastases than one that has been present only one month.

The grading is of some importance in evaluating *radiation sensitivity* for the less differentiated growths seem to melt away under this treatment almost miraculously at times. The only drawback is that they also are more apt to

"freeze up" again than a low grade tumor that has shown primary healing. The subject of radiation sensitivity cannot be discussed here, however.

It is extremely encouraging to obtain primary healing after removal of carcinomata. Although it is welcomed as the best possible sign, it does not prove a cure. Cures should not be reported just because primary healing has occurred. A definite time limit of three to five years without recurrence is the accepted criterion of cure, but one can report "arrest" of the growths for any period.

Multiple Carcinomata. These do occur, and a person who has had one carcinoma is three or four times more likely to have another than had he not had the first. The mucosa and skin seem to have a tendency to "wear out" in certain patients. Such individuals should be watched very closely, at least two or three examinations a year should be made. Some of the so-called multiple carcinomata may, of course, be late recurrences of the earlier growths. Trotter has offered a theory that there is possibly some natural immunity to cancer and that after treatment, the host is thus free for a time of evidence of growth, but that any part left behind may slowly grow and finally overcome the immunity the host carries.

Radiation burns may occur in an area of growing carcinoma and the two may be very difficult to distinguish. The lesion may be all radiation slough or ulcer, or this plus more cancer. The only way to be certain of the diagnosis is by biopsy or complete removal of the lesion and examination of the tissue.

Cancer pastes continue to be popular and older people seem especially susceptible to their charm. A corollary is that possibly the surgeon's treatment is not well thought of. However, cancer often is found right in the area of a paste slough. The process of destruction by paste is generally more unpleasant, especially if bone is involved, than the general public is led to believe.

Sarcomata. In older people sarcomata of the jaws are not so frequent as in younger patients. They are of the osteogenic type or of the somewhat less dangerous type designated as ossifying fibromata. These tend to recur following removal and develop into a true osteogenic type. Ossifying fibromata may be successfully resected occasionally, but treatment of the osteogenic type is of little final avail.

Benign Tumors. These occur more often in old than in young persons.

Leukoplakia is the most important benign tumor. It occurs as a flat lesion that can hardly be distinguished as a tumor. If ulceration or wart like verrucous growths appear in one of these areas, however, then the lesion is definitely a cause for alarm and should be removed promptly. The most efficient method is surgical removal, either by diathermy, cautery, or excision and suture. Because of the differentiation of the cells involved, radiation is not commonly recommended. On the other hand, if the mouth is so full of the disease that total excision is impossible, interstitial radiation or contact x-ray therapy can be applied. If even a partially satisfactory result can be obtained, surgical attack can be made later. It is in the treatment of these widespread leukoplakias that the worst radium burns are likely to occur. They cause pain which is almost unbearable. The only effective treatment is wide surgical removal with immediate skin grafting, done under general anesthesia.

The care of a patient with a tendency to leukoplakia includes thorough cleaning of the mouth in every way, repair of dental caries, limitation of tobacco, frequent use of a mild mouth wash, such as 5 per cent potassium chlo-

rate in alkaline antiseptic, and the use of a soapy tooth paste. It is very important to remove surgically any areas that tend to change their character, the significant changes being, as stated above, ulceration or extensive thickening of the plaques.

Precancerous lesions are hard to define and the term is used loosely. Leukoplakia unquestionably is the most important precancerous lesion. There has been a good deal of discussion of the relation of ulcers around teeth, ulcers opposite carious teeth, irritations due to ill-fitting plates and bridge work, to the occurrence of oral cancer. Some authors have suggested that dental caries is the cause of oral carcinoma. There seems to be little point to many of the arguments. More important is the concept that it is wise to recommend the relief of all irritations in the mouth, whether one suspects them of being precancerous or not, simple cleanliness, operative removal, and microscopic examination of any lesions that do not clear up.

Fibromata of the alveolus occur in some older edentulous patients, especially in the anterior upper region. The cause is unknown. The growth often continues to grow and causes very ugly dirty folds that encroach upon the lip and make the wearing of dentures impossible. These should be treated by a wide excision with immediate repair. If recurrences appear, they should be removed before they cause too much deformity.

Torus palatinus is not a lesion limited to the elderly, but is often first noticed in age because of some irritation or inability to fit dentures over the deformity. The process is a peculiar slow-growing bony overgrowth in the mid line of the palate. It has been observed in patients as young as ten years of age. It is usually symptomless unless the overlying mucosa is injured or irritated. Treatment is by excision, after reflecting the mucosa. Rarely is it necessary to remove the bony growth, however.

Hypertrophied circumvallate papillae occur frequently on the tongues of older patients. There is considerable pain. The common term, "painful tongue," is almost worthless as a diagnosis, but almost no further information seems to be available on the subject. The occurrence of the pain cannot be denied. There is no known successful treatment. Frequently these patients suffer from a phobia, often fearing cancer. Mild sedation and reassurance to relieve the fear of cancer usually produce fairly good therapeutic results. Cauterization, dissection, removal, or strong chemical applications to the papillae should not be done. As little attention should be drawn to the area as possible.

VITAMIN DEFICIENCIES

Vitamin A. Keratinization and hyperplasia of the gums are not uncommonly found in a fairly advanced deficiency. *Treatment*, as in any vitamin deficiency, should consist of a well balanced diet in addition to an abundance of the particular deficient vitamin. The minimum allowance is between 3000 and 6000 units daily, but even greater amounts around 25,000 units daily are required for treatment. The difficulty of absorption in the debilitated should not be overlooked.

Vitamin B. Deficiency in the B complex causes angular stomatitis and glossitis. Cheilosis, or angular stomatitis, is the presence of superficial transverse fissures exactly at the angle of the lips, usually on both sides with very little inflammatory reaction, caused by a lack of riboflavin (B_2) in the diet. The tongue assumes a purplish red color. Ariboflavinosis may result from a

poor diet, faulty absorption, or poor utilization as in liver damage *Treatment* is regulated as to the cause, *i e* , if there is poor absorption or utilization 10 to 20 mg crystalline riboflavin should be given daily by mouth, otherwise, only 3 to 5 mg a day (See p 191)

The ' beefy looking ' red smooth, dry tongue of pellagra is the other important change noted in the mouth in vitamin B deficiency This is due to desquamation of the superficial epithelium and may vary from red patches and vesicles to ulceration of the tongue and mouth as in sprue Pellagra is due to the B group avitaminosis, predominantly nicotinic acid The *treatment* consists of 200 to 500 mg orally or 40 to 80 mg intravenously of niacin with 200 to 250 mg of powdered brewers' yeast daily in divided doses (See p 191)

Vitamin C. The interdental papillae and gingivae show enough change in vitamin C deficiency that macroscopic and microscopic examination offer a simple method of detecting the lack as described by Kruse In the acute phase there is a shiny, red, congested appearance of the gums, not uncommonly with bleeding and infection This may proceed to frank scurvy, ulceration, and necrosis Less severe further progression leads to a chronic phase of atrophy *Treatment* consists of the addition to the diet of large amounts of oranges, to matoes, carrots, and leafy green vegetables If these cannot be tolerated or if outspoken scurvy is present, ascorbic acid in crystalline form should be given orally, or 200 to 500 mg of a properly neutralized aqueous solution should be given intravenously daily

INFECTIONS OF THE MOUTH

Tuberculosis of the tongue occurs occasionally It seems not to occur primarily, but always in association with pulmonary involvement (see p 324) There is a large deep dirty, and hard ulcer that is usually very painful Local treatment may be by complete diathermy excision and/or the application of interstitial gold radon seeds

The tertiary lesions of *syphilis* are rare in the aged but do occur Gum mata of the tongue are not so exceptional as in younger persons They cause a foul, discharging ulcer, the progress of which is entirely destructive, with no new tissue piled up at all The edges of the ulcer are swollen The absence of marginal growth distinguishes the luetic lesion from carcinoma No local treatment is necessary Systemic antisyphilitic therapy is of course indicated Complete healing of the ulcer may occur after a single arsenical injection The first stage of syphilis is very rarely encountered in the aged

Noma is a *gangrenous stomatitis* that is not a primary disease in itself, but an expression of some other debilitating disease, such as a blood dyscrasia or

ice X ray therapy is of questionable value The old idea of burning the area now has but very few advocates The lesion is so rare as hardly to warrant description It is important, however, to try to find the primary cause when it does occur There may arise misconceptions as to cause such as that some innocent dental trauma, which happened to be coincidental, is responsible

Stomatitis, in all its various forms, can occur in older persons but none

of the primary disorders is limited to the elderly. Pellagra, scurvy, the anemias, Vincent's infections, and so on occur at all ages. The Vincent organisms have been credited with being the cause of much oral infection. Whether this is true or not the mouth should be kept as clean as possible throughout life. Soap will control the Vincent's organism about as well as anything. Therefore the use of soapy tooth paste and of hydrogen peroxide or sodium perborate should be advised promptly on slightest suggestion of trouble.

Ulcerative stomatitis, due possibly to a virus, the common canker sore, or herpes, may cause such intractable pain that surgical section of the lingual nerve may be indicated. Otherwise, medical care and cleanliness of the mouth are indicated. In severe episodes, morphine and local anesthetic drugs may be needed.

TEETH

Root Abscesses. *Gum-boils* are root abscesses that form alongside a tooth. The abscess may present itself lower down in the buccal fornix. They are to be opened, simple hot irrigations should be used until the cellulitis subsides and then, in ten to fourteen days, the tooth usually can be removed safely. In an older person, there is seldom any point in trying to save a tooth that has a draining abscess on its roots. One extremely important point is not to pull every infected tooth during the acute stage of the abscess, for there is already a localized osteomyelitis, the additional trauma of the extraction, instead of just relieving the pus, may produce a fully developed osteomyelitis, or a spread of the infection into the soft tissues.

Dental Root Cysts or Chronic Dental Root Abscesses. These, around the roots of dead teeth, have been widely and variously charged with the cause of all sorts of ailments elsewhere in the body. The concept of focal infection is too well known to require discussion here. There are different opinions as to the importance of such root infections, but the uncertainty is immaterial since it is so easy to get rid of these teeth, and since they are no good as teeth anyway. There does not seem to be much argument against removing definitely infected teeth if there is other trouble or any suspicion of focal infection. The main difficulty in deciding on the removal of teeth has been with those not radiographically bad. This uncertainty will probably continue. We tend not to count upon much improvement following the removal of most "bad" teeth.

Pyorrhea Alveolaris. While not limited to old patients, this, of course, is oftener found in those of mature years. It occurs in persons with sound teeth as well as others. Many different modes of treatment have been advocated, but all are of little avail up to the present. The essential therapeutic measures are cleanliness and drainage of the pockets under the gums.

Other Affections. *Osteomyelitis, actinomycosis, and tumors of the dentigerous bone*, such as adamantinomas, all occur in older persons, but are not specific to them. *Neck infections in the internal pterygoid region* are serious accompaniments of dental infections and have to be dealt with frequently, using chemotherapy and adequate external drainage.

Giant Cell Tumors. Although these occur infrequently, when recognized, general metabolic studies, including blood calcium analyses and x-ray ex-

Ludwig's Angina. This septic phlegmon of the floor of the mouth is encountered often in older patients and is treated with chemotherapy, x-rays, and open surgical drainage. The idea that pus is not present in these lesions is wrong, for it invariably exists in some small pocket. The pockets are often missed by making only small incisions and shallow explorations of the affected area.

Antral Fistulae. Such fistulae frequently occur through old tooth sockets and often require multiple operations for closure. If the discharge is bad enough, or if food gets in the antrum, closure should be done by cleaning out the whole antrum, removing the medial antral wall into the nose (for drainage), and then repairing the fistula with mobilized cheek and palate flaps.

Unerrupted Teeth. Especially when in the upper jaw, unerupted teeth may occasionally give trouble in older persons, by causing irritation or chronic infected draining sinuses. They should be removed.

Dental Neuralgia. It is extremely important to differentiate *dental neuralgia* from *tic douloureux*. The first is usually a constant boring pain, although, if due to a pulpitis in a dying tooth, it may simulate a tic almost exactly. The distinguishing points are that the pain of *tic douloureux* lasts longer, there is no trigger point, the patient often cannot even tell in which jaw the pain is located (upper or lower), and it is usually set off by cold applications. The neuralgia that comes from a crushed nerve canal in the lower jaw, a malignant growth, a deep infection, or a neuroma of a superior dental nerve is usually constant and boring. The patient may become morose, may quit work entirely, and may even remain depressed after the pain is relieved. Many teeth are needlessly pulled when the trouble is an outright *tic douloureux*, but if the trouble does happen to be one of the above mentioned dental neuralgias that some simple procedure will relieve, it is not logical therapy to cut the whole nerve to the area as in a tic operation (see p. 289).

Dental Caries and Dentures. For caries, or decay of teeth, in the aged, nothing can be done other than restoration or removal. There is a tendency for older people to want to get rid of all their teeth, if they have had a good deal of dental trouble, but it is the distinct feeling of this author that at least the anterior teeth should be left in as long as possible, even if dentures have to be built around them. This applies especially to older women.

Dentures should be removable ones in older patients. They are cleaner, usually more comfortable, and do not damage remaining teeth as much as do fixed bridges.

Auriculotemporal Pain Syndrome. There is an occasional patient who complains of intractable pain about the ear, temporal region, or even down into the tongue, that may be dependent on an erosion of the temporomandibular joint and irritation of the auriculotemporal nerve. This is a difficult diagnosis to make and many misinterpretations are possible. The syndrome is apt to occur in patients who have no molar support, i. e., no molar teeth in apposition, or in patients who have worn the same denture for years and have lost a good deal of the molar space from absorption. A useful diagnostic test is to see if the pain is relieved by opening the jaw or swinging it from side to side, or by supporting it wider open with rubber plugs held between the molars. X-rays of the joint surfaces help somewhat in the diagnosis. If the diagnosis is established, relief can sometimes be obtained with new dentures which have a wider molar space, or by building up any remaining molars.

Temporomandibular Meniscus Syndrome. This depends upon a derangement of the meniscus. This may be acute, from dental trauma in a difficult extraction, or external trauma, or an old progressive derangement. There are three symptoms "cracking" or popping, pain, and blockage. They usually appear in this order, but may vary. Treatment is by rest, voluntary on the part of the patient in the use of the jaw, use of chin-vertex bandages, or wiring the jaws in occlusion. Injection of the joint capsule and removal of the disk are hardly ever indicated in older patients.

Fractures of edentulous lower jaws present problems of fixation. They may be held by direct wiring through drill holes made in the fragments, fixing the lower dental plate in place with wires around it and the jaw, or by bandaging the chin to the vertex with the dentures in place. In some instances, an internal wire driven across the fracture line with a power drill may be the treatment of choice and will allow open mouth treatment. The jaw may be so atrophic, because of an edentulous condition persisting over a long period of time during which heavy plates have been worn, that only the thin bottom edges remain. Under these circumstances no direct method of fixation of the fracture may suffice and a bone graft may be necessary.

SALIVARY GLANDS

Salivary Stones. These may occur at any age and may have to be removed, either through the duct or from the gland. Stones in the submaxillary gland may require removal of the whole gland if the stone cannot be dislodged. Such rigid fixation of calculi is very rare in the parotid gland. The parotid is much less frequently involved by calculus formation.

Inflammation and Catarrh. Chronic inflammation of the salivary glands may exist without stones and a catarrhal blockage of the saliva flow occurs frequently. This occurs more often in the parotid. When the gland is once infected it is somewhat comparable to an infected prostate. Massage and probing of the duct is usually sufficient treatment. It is important to avoid surgical drainage of the duct through the face as to do so may result in the formation of a permanent fistula.

Salivary Gland Tumors. *Mixed tumors of the salivary glands* occur in older persons and should be removed surgically. If the tumor become malignant, the seventh nerve may have to be sacrificed, but gold radon seeds can be used with some success. X-rays do not seem to have much effect on these tumors. Tumors of the submaxillary glands have a higher percentage of malignancy than those of the parotid and as they are prone to extend through the floor of the mouth, interstitial gold radon seeds are usually relied upon in treatment in preference to surgical excision.

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CHAPTER 33

DISEASES OF THE ESOPHAGUS AND STOMACH

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ESOPHAGUS

In elderly people diseases of the esophagus are encountered infrequently, but any lesion involving its function is apt to be of a serious nature. Ordinarily this tube is 25 cm long and from 1.6 cm to 2.2 cm wide. The mucous membrane consists of stratified squamous epithelium, muscularis mucosae, flexible connective tissue, and scattered islands of mucous glands similar to those found in the stomach. Motor activity is furnished by an inner circular and an outer longitudinal layer of muscles.

Acute Esophagitis. Mechanical trauma, chemical and thermal irritation may produce an esophagitis. Acids, alkalis, or other poisons result in severe injury, especially at the points where the esophagus is narrowed and at the lower end where the rate of passage is slowed. A moderate to severe esophagitis may be produced by infectious diseases such as diphtheria, scarlet fever, smallpox, typhoid fever, syphilis, by secondary inflammation from swallowing infectious material from the respiratory tract, mediastinal diseases, and tumors in or near the tube.

Symptoms. Where irritation is marked, there is pain along the course of the gullet aggravated by swallowing, extreme thirst and expectoration of blood-streaked mucus may occur. Passage of tubes is painful and not advisable until the acute phase has passed and a fluoroscopic examination has been made to eliminate the presence of foreign bodies or anatomic defects.

Treatment. Anesthetic solutions are often sufficient in the less severe forms of esophagitis. Appropriate antidotes are useful immediately after poisoning but are usually of little value by the time the patient is seen. Warm milk and water, given frequently and in small amounts, are tolerated well. Spasm may interfere with swallowing and tubes may have to be passed, but this should be done with caution. Intravenous infusions may be necessary to prevent dehydration. Every patient with acute esophagitis should be observed over a long period of time because a stricture may develop.

Chronic Esophagitis. Chronic esophagitis is very unusual except following chronic esophageal stasis such as results from stricture, cardiospasm, regurgitation, or prolonged use of tubes. Removal of the source of irritation usually results in prompt relief. Resulting strictures should be dilated frequently for a long period of time. Metal olives are useful, but French bougie dilators are preferable. These are passed over a wire which has been guided over a silk thread anchored in the intestines. Such dilatation may have to be carried out at frequent intervals for several years.

Ulcer. Ulcer is not uncommon in persons who have suffered a long exhausting disease such as carcinoma, tuberculosis, or typhoid fever.

Pressure ulcers occur from encroaching tumors outside the esophagus.

and may be found at any level. Continuous tension produces local anemia, secondary infection, and later sloughing of dead tissue.

Uremic ulcers are seen in severe nephritis and are usually symptomless.

Peptic ulcer is most frequently found in the lower end of the gullet under conditions similar to gastric and duodenal ulcer. There may be regurgitation of the gastric juice, increased abdominal pressure may facilitate regurgitation. The lesion appears grossly and microscopically similar to a gastric or duodenal ulcer and may extend through the cardia into the stomach. Perforations into the mediastinum as well as hemorrhage have been reported. The ulcer may heal to form a scar and produce a stenosis.

Symptoms These ulcers produce dysphagia and pain referred to the site of the lesion. Pain under the xiphoid process relieved by food or alkali is characteristic of peptic ulcer.

Treatment Soft warm liquid food and occasional swallowing of warm olive oil bring about most relief in upper esophageal ulceration. Esophageal ulcer is best treated in the same manner as gastric and duodenal ulcer. Gastrostomy may be necessary if stenosis occurs.

Spasm Spastic contraction of the esophagus may occur at any point. It is usually located at the cardiac sphincter (cardiospasm) and may be transient or chronic and give rise to symptoms of obstruction with regurgitation of food and mucus. When chronic, there is often marked dilatation of the gullet above the spasm as much as 2000 cc. of food and liquid may be retained.

Certain affections of the central nervous system, such as tetanus, "hysteria," chorea, epilepsy, and hydrophobia, may produce esophageal spasm. Reflex phenomena from disease of some distant organ such as ulceration of the larynx and gastric or peritoneal disease, have been accused. The idiopathic form probably due to a fault in the relaxation mechanism or increased sphincter tone, is most common.

Pathology Obstruction due to cardiospasm causes retention and stagnation of food which is followed by dilatation and hypertrophy of the walls. Decomposition of retained food irritates the esophageal lining and chronic catarrhal inflammation with an abundant secretion of thick glairy mucus results. Unless relieved, severe dehydration and starvation will occur.

Symptoms The clinical picture varies with the degree of esophageal obstruction. There may be substernal and epigastric distress with great dilatation and an entire meal may be taken before distressing symptoms appear. True vomiting is not present. The patient usually complains of vomiting, but nausea and retching are absent and the material is regurgitated. Liquid and solid foods are taken with equal difficulty. Not uncommonly mucus drains from the mouth during sleep. Regurgitated material may contain food eaten twenty-four hours previously.

Physical Findings An olive-shaped bougie may be arrested temporarily at the site of the spasm or it may slip through without the slightest effort. The constriction is usually found at the cardia, 16 inches from the incisor teeth.

X-ray examination after swallowing thick barium shows a fusiform dilatation above the point of spasm.

cover which limits expansion to a circumference of 4 to 6 inches. A second rubber bag is fitted over the silk casing to facilitate passage through the gullet. Inflation of these bags is accomplished through a rubber tube connected to the inner bag. In most instances it is safest to guide the instrument over a silk thread which the patient has swallowed twenty-four hours previous to dilatation. The distance between the incisor teeth and spasm is then determined with a large bougie, a piano wire passed over the string and the dilatation bag passed over the wire so that its midsection lies at the point of constriction. A manometer is placed in the inflation system, pressure of 250 to 300 mm. mercury is quickly reached and maintained for about five seconds. Then the bag is deflated and the instrument withdrawn. After this procedure food is passed into the stomach without difficulty.

Diverticula. Diverticulum of the gullet is not infrequent in later life. Pressure, or pulsion, and traction diverticula are found. The *pressure diverticulum*, as its name implies, is due to long continued pressure within the esophagus. It occurs chiefly at the narrowest portions of the tube, usually at the cricopharyngeal level, only occasionally at the bifurcation of the trachea, and infrequently just above the diaphragm. The most probable causes are defective fusion of the muscle bands, or long periods of pressure over fibrous tissue with a resultant outpouching. The affection develops slowly and is usually found after the age of forty-five years.

Traction diverticulum is more common and is caused by pull from without the esophageal wall. Diseases producing a cicatricial contraction, such as tuberculous lymph glands and pulmonary inflammatory processes, are the most common causes. As a result, such diverticula are seen chiefly at the bifurcation of the trachea. They may be single or multiple, are usually small and triangular in shape, with the apex toward the source of pull, and are symptomless.

Symptoms. The onset is gradual with a sense of stoppage of food and liquid above the stomach. Frequently the first complaint is of regurgitation of a small amount of liquid during sleep, the material consisting chiefly of glairy mucus without hydrochloric acid or gastric ferments and thus easily differentiated from gastric contents. Decomposition of sac contents may take place, causing a foul odor to the breath. This in turn is followed by substernal burning, pain, and a feeling of the presence of a foreign body in the throat. The appetite is usually good but loss of weight occurs when insufficient food is retained.

Physical Findings. Commonly if an attempt is made to drink water rapidly, a characteristic gurgle is heard as the patient swallows. If the sac is in the cervical portion of the esophagus and is full, it may be seen or palpated, usually on the left side of the neck.

X-ray examination will disclose the characteristic outpouching when thick barium is swallowed.

Treatment. Small diverticula which cause little or no difficulty are best left untreated. In large defects, where the symptoms remain distressing in spite of high-calorie, soft dietary management, surgical removal may be necessary. Tube feeding to build up the patient's strength should precede any operative procedure when there has been starvation. If the sac lies in the cervical portion of the esophagus removal is facilitated. Certain thoracic diverticula can be removed but frequently the approach is so difficult and the mortality so high that a gastrostomy or frequent periods of tube feeding are preferable.

Tumor Obstruction may result from both intra and extra esophageal growths or inflammation. This condition is characterized by stoppage and regurgitation of food or liquid shortly after swallowing, difficulty in passing a



Fig 103 Lateral view of a pulsion diverticulum of the esophagus



Fig 104 Anteroposterior view of the same pulsion diverticulum shown in Fig 103

stomach tube or bougie and x ray findings of obstruction. Frequently there is some esophageal dilatation above the constriction and large amounts of glairy mucus are regurgitated.

Tumors of the thyroid gland, especially if substernal, may cause compression. Aneurysm of the aorta, mediastinal new growths or abscesses, enlargement of, or cicatrix following the healing of, infected tracheal and bronchial lymph nodes, diseases of the vertebrae, laryngeal tumors, pericarditis with much effusion and great dilatation of the right side of the heart have been found to produce varying degrees of obstruction.

Of the intra esophageal causes benign tumors, such as fibromata and papillomata, are very rare. Impacted foreign bodies are unusual. Diverticulum and gumma should be kept in mind. Carcinoma, unfortunately, is the most common cause of intra-esophageal obstruction in patients past middle life.

Carcinoma Symptoms Unless an early lesion is found during routine fluoroscopy, the patient is rarely aware of the growth until signs of obstruction occur. In persons over fifty, persistent difficulty in swallowing accompanied by rapid emaciation usually indicates esophageal cancer. First, solid foods like bread or meat cannot be swallowed, then soft foods and finally liquids are taken with difficulty. Regurgitation of food and mucus from the esophagus occurs. Frequently a small amount of decomposed food, blood, and pus may be brought up and a foul breath develops. A feeling of pressure below the sternum is noted and is often associated with pain. Later pain may be lancinating, especially after lying down at night. In spite of marked emaciation, hunger is rare, thirst is common. Cachexia and severe anemia appear as the disease advances.

Physical Findings Extra esophageal tumors can sometimes be palpated in the neck. It is much safer to examine the esophagus by fluoroscope before passing a tube, especially if obstruction is suspected. Benign tumors may occur in any portion but carcinoma occurs most frequently in the lower half. Intra-esophageal tumors can be observed through the esophagoscope and biopsies made to determine their nature.

Treatment Treatment of extra esophageal compression should be directed at the primary cause. If this cannot be relieved, a liquid, high calorie, high vitamin diet should be prescribed. Benign tumors and foreign bodies can frequently be removed through the esophagoscope. Carcinoma grows slowly and attempt at removal of an early lesion is justified. X ray treatment as a rule has

is guided by a string anchored in the intestines. A channel may be kept open and nutrition maintained for many months by this means. The procedure is not uncomfortable and can be done frequently, anesthesia is seldom needed. Later a gastrostomy may be imperative.

Perforation This complication may result from an ulcer, trauma from instrumentation, a foreign body or forcible swallowing when the wall has been weakened by a previous injury, and the elasticity lost due to scar tissue formation. We have seen two patients with old lye strictures who experienced ruptures, one following a meat bolus which the patient attempted to force through by swallowing water. The second patient experienced perforation after a se-

Treatment must be promptly and energetically instituted. Morphine to

control pain and stimulants to overcome collapse are necessary. All fluid and food should be withheld by mouth for some time. Protein, carbohydrates, fluids, and electrolytes to suit the needs can be given by vein. A blood sulfonamide level of at least 10 mg per 100 cc should be maintained (sodium sulfathiazole). In addition, 100,000 units of penicillin every three hours should be given by hypodermic. Streptomycin may or may not be useful. Trans-thoracic surgical drainage in selected cases may be life saving.

Granuloma This is rare and if present is usually masked by disease elsewhere. Gumma must always be considered in a patient with an esophageal tumor and with a positive complement fixation reaction. Tuberculous involvement is secondary to pulmonary infection. Blastomycosis, actinomycosis, and similar conditions are very rare.



Fig 105



Fig 106

Fig 105 Perforation of esophagus following severe coughing in a patient with an old lye burn.

Fig 106 Scarring of esophagus from lye. Same patient as shown in Figure 105 eight months after perforation.

Varices Tortuous dilatation of the esophageal veins almost always occurs in the lower end of the tube. Patients with portal obstruction as seen in cirrhosis of the liver and Banti's diseases are subject to this complication. These vessels are easily traumatized by swallowing, and repeated severe hemorrhages or marked anemia may be the only evidence of their presence. Frequently it is very difficult to visualize them under the fluoroscope and esophagoscopy may be the only means of determining their presence. If they are suspected, all esophageal instrumentation should be done with great caution. Treatment is symptomatic; reduced blood volume seems to favor less frequent hemorrhages and quicker recovery after rupture. Transfusions may be necessary. Prothrombin estimations and an adequate vitamin K level should be maintained. Injection of sclerosing solutions by means of the esophagoscope has been used.

with some success. In severe hemorrhage pressure locally over the ruptured vessel by means of an inflated bag is advocated.

Neuroses. Neuroses are usually associated with the general picture of neurasthenia and/or "hysteria." Normal esophageal sensory perception is almost absent and transient motor abnormalities can rarely be demonstrated. Treatment should be directed toward the underlying cause. Often larger doses of sedative are required than in similar instances without esophageal manifestations.

STOMACH

Physiology and Anatomy. The functions of the stomach are secretory, motor, and absorptive. Absorption is negligible since only a little alcohol, water, and a few peptones are taken up. Three muscular coats provide motor activity, the tone in these muscles prevents food from dropping directly from the cardia to the pylorus, causing the stomach to fill from above. An ordinary meal should leave the stomach within seven hours.

Both an external and internal secretion occur. Little is yet known about the internal secretion, most gastrointestinal hormones discovered to this time seem to be formed in both the stomach and intestinal tract. Ordinarily about 1500 cc. of clear gastric juice is secreted in twenty-four hours. The chief constituents of this juice are water, hydrochloric acid, pepsin, rennin, and mucus. The concentration of hydrochloric acid as secreted by the parietal cells is 0.17 normal, constant, and isotonic with blood, lesser concentration of acid is due to dilution and buffer substances. Pepsin and rennin are formed by the chief cells. Secretion is an almost continuous process, usually 10 to 30 cc. or even 50 cc. may be found in the fasting stomach. The maximum flow occurs thirty to forty-five minutes after eating. A substance known as the intrinsic factor of Castle, essential to maturation of red blood cells, is also secreted in the normal stomach.

The physiology of the 'pyloric sphincter' is still obscure. At autopsy this opening may admit three fingers but during life the orifice is about one-third that size. Opening and closure take place rapidly and the gastric contents are squirted into the duodenum in jets. Probably the pH of the gastric and duodenal contents may influence the action of this valve to some extent.

The nerve supply to the stomach is derived from the vagus and the splanchnic branches of the sympathetic from the posterior root ganglia. The sensory nerve supply is through the sympathetic fibers.

Functional Disturbances. Many systemic disturbances produce epigastric discomfort and the stomach is thought to be at fault. Strong contractions of the walls of the stomach can occur with hunger, appetite stimulates secretion of salivary and gastric juice. Most of these reactions are the result of the physical and psychic state rather than of any condition within the stomach itself. Excessive hunger or craving after certain foods is due to psychogenic or endocrine disturbances. Loss of appetite may also be emotional, due to debility or to endocrine imbalance. Unhappy living conditions, a monotonous or deficient diet, or a continued nervous tension will frequently reduce the desire for food.

Vomiting, when no organic pathology is present, is generally due to emotional instability. It may assume the form of regurgitation or actual vomiting. Pain, distress, and nausea are absent. Loss of weight is unusual even when

vomiting has been present for many years. Liquids are usually vomited more readily than solids. This condition is frequently a defense mechanism or due to a false belief that an organic disturbance is present.

Fermentation and gas formation within the stomach is rare and almost all "belching" is due to swallowed air. Air may either be ingested while eating hurriedly or swallowed unknowingly throughout the day. This process can be demonstrated under the fluoroscope. Reduction of nervous tension with mild sedatives and removal of sources of irritation usually produce prompt relief.

Abdominal migraine is rare in the later years.

Motor Disturbances. Dysfunctions of the gastric musculature are not common. Retention is usually due to an organic obstruction. With gradual stoppage of the gastric outlet, marked hypertrophy of the walls with hypertonicity rather than atony occurs.

Acute dilatation occurs following a severe injury or after an abdominal operation. Gastric and duodenal secretions as well as food are retained and as much as two quarts or more may be vomited or aspirated. Continuous aspiration through a small rubber tube should be employed until the obstruction is relieved. Complications such as aspiration pneumonia, dehydration, or chloride deficit due to withdrawal of large amounts of hydrochloric acid should be kept in mind.

Gastric spasm occurs locally at the site of an ulcer. It has also been described in the gastric crisis of tabes.

Secretory Disturbances. The normal gastric secretions vary so widely from hour to hour and from day to day that it is very difficult to set a standard. An indication of the degree of acidity may be obtained with the Ewald or any other test meal. Occasionally histamine or alcohol test meals are necessary and even with such stimulants varying results are obtained. From 4 to 10 per cent of normal people have no demonstrable free hydrochloric acid in their gastric juice and most of these suffer no ill effects. It is important to differentiate achlorhydria from achylia. Achylia can be determined by the lack of free acid and failure of the juice to coagulate milk. In older individuals there may be a physiologic decrease in the amount of acid secreted. Rafsky and Weingarten¹ found an incidence of 23.5 per cent of normal people over sixty-five years of age with no free gastric acid on a bread and water test meal, 8.5 per cent of these were false because histamine stimulated a flow of acid, ferments were present in all instances. Some patients without an acid secretion are benefited by 10 to 15 drops of 10 per cent hydrochloric acid in a glass of water taken with meals.

High or low acid values as obtained in one or two tests do not necessarily indicate hypo- or

High acid values

gastritis. Increase

many patients with ulcer, especially if pyloric obstruction is present. Such a secretion frequently continues throughout the night until the obstruction has been relieved.

Diverticulum. This is rare and consists either of an outpouching with all the layers of the stomach wall present or, more commonly, a herniation through a thin or fibrous portion of the stomach. Symptoms are usually absent and the diagnosis is made incidental to an x-ray examination. Unless complicated by inflammation it is best treated symptomatically.

Diaphragmatic hernia may result from failure of the leaves of the diaphragm to unite or from abdominal trauma which separates a weak union between the muscles. No gastric defect is present except when there is a shortening of the esophagus or a pinching off of a portion of the stomach. Distress results from obstruction and ulceration may occur. In most instances no treatment is necessary and in some, caution against eating large meals and lessening of intra-abdominal pressure by weight reduction is sufficient. Calcium carbonate or a glass of milk is often sufficient to control the occasional symptoms. If severe irritation and ulceration are present, a peptic ulcer type of treatment may be useful. Repair of the diaphragmatic defect is indicated when it is large and symptoms remain distressing.

Specific Inflammations. Foreign Bodies Astonishing reports of foreign bodies such as glass, nails, open knives, and the like being swallowed without harm have been made. Many such articles are passed through the intestines but some remain in the stomach. Bezoars may form from continued hair swallowing or from insoluble precipitates of certain foods such as persimmons. Any foreign body retained in the stomach will produce symptoms of irritation with epigastric burning, nausea, and vomiting. They can usually be seen by x rays and are best removed surgically.

Corrosives Strong alkalis or acids will cause a severe burn or perforation of the stomach wall. Immediate lavage with an appropriate antidote is most useful. Drinking water and induced vomiting are also useful measures. Treatment is symptomatic and nutrition and fluid balance can be maintained temporarily by intravenous infusions. Blood transfusions may be necessary. After the acute stage has passed, frequent small feedings of liquid or soft foods may be given as tolerated. If a solid, such as a caustic stick, has been swallowed, an hour glass type of deformity due to a localized injury may result. When severe pyloric obstruction results a gastroenterostomy is necessary.

Excessive use of alcohol causes varying degrees of acute gastritis with symptoms of epigastric burning, discomfort, and even vomiting of blood streaked mucus. Chronic alcoholism would seem to keep such an irritation active, yet there is some doubt, both from postmortem and gastroscopic studies, whether this is true.

Phlegmonous Gastritis This condition is very rare and uniformly fatal. A diffuse inflammation of the walls of the stomach is said to occur as a result of bacterial invasion through abrasions or carcinomatous ulcerations. The symptoms are those of sudden severe epigastric pain, abdominal rigidity, and shock. The diagnosis is usually confirmed at operation.

Limitis Plastica or Scirrhus Gastritis Low grade inflammation was thought to result in the unusually thickened small leather bottle type of stomach. Later study has shown this change to be due to a slow growing scirrhus type of carcinoma. Treatment is similar to other forms of gastric carcinoma.

Syphilis Secondary lesions characterized by mucus patches on mucous membranes probably account in part for the abdominal discomfort and lack of appetite seen in this stage. Gumma is unusual and is difficult to differentiate from gastric cancer. There is usually a contraction of the body of the stomach about the lesion and ulceration at its center. Pain may be gnawing, persistent, and not necessarily relieved by food or alkali. Frequently free hydrochloric acid is absent and the complement fixation test positive. Vomiting, weight loss and anemia may be quite severe. Antisyphilitic therapy gives spectacular but

limited improvement. If there has been much scarring a contracted or hour-glass stomach will result.



Fig 107

Fig 107 Syphilis of the stomach



Fig 108

Fig 108 Same patient as shown in Figure 107 after five years

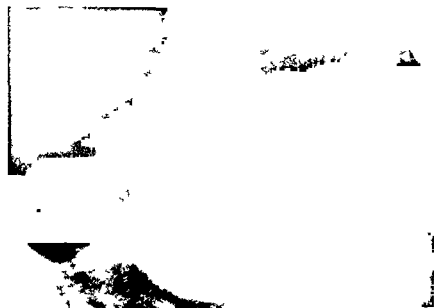


Fig 109 Same patient as shown in Figure 107 after seventeen years

Tuberculosis Gastric tuberculosis is rare and almost always secondary to a pulmonary focus. Attention should be directed to the primary infection since little can be done for the gastric lesion.

Lymphogranulomatosis, Blastomycosis, Actinomycosis, and Other Affections These may be primary but are usually not discovered until other organs are involved. X ray therapy seems to be useful in lymphogranulomatosis. The usual measures in treating actinomycosis and other mycotic affections should be tried.

Gastritis Acute This condition, due to the ingestion of irritating food or infection, was first seen by Beaumont and has since then been observed through the gastroscope. The mucous membrane becomes reddened with patches of intense hyperemia, an excessive amount of mucus is secreted, and even papular elevations may be seen. These promptly disappear when the source of irritation is removed. The diagnosis can often be made from the history.

Chronic The classification proposed by Schindler—superficial, atrophic, and hypertrophic gastritis—seems most practical. Inflammation may occur in localized patches or be diffuse, it most frequently involves the body of the stomach.

In *superficial* gastritis reddening of the mucous membrane, edema, and exudation, with superficial erosions and submucous hemorrhage, may occur. Generalized epigastric distress and tenderness with a history of excesses in eating and drinking are usually present. Gastric analysis and x ray studies are inconclusive. Treatment is symptomatic with correction of the diet.

Atrophic gastritis is characterized by a thinning of the mucosa chiefly in the body of the stomach. Complete atrophy is unusual. The mucosa is gray or greenish-gray and branching blood vessels are seen through it. The normal gastric folds are decreased in size or absent and frequently the papillae of the tongue are also flattened. Anacidity or low acid values are the rule and x ray studies show no changes. Anemia of the pernicious or of the hypochromic type may be present. Treatment with liver or iron and correction of the deficiency will frequently improve the appearance of the gastric mucosa but return of secretion of hydrochloric acid is unusual.

Hypertrophic gastritis may be patchy or diffuse, with a dull velvety mucous membrane and irregular thickening of the rugae. Progressive thickening may cause duplication of folds to give a warty appearance. Superficial ulcerations are common. Exacerbations and remissions occur frequently. Generalized epigastric distress, often of a periodic type as seen in ulcer, is usual. Treatment comprises removal of irritation, frequent small meals or a modified type of antacid ulcer management is usually satisfactory. No characteristic gastric acidity is found but x-ray examination often reveals the thickened folds. Localized areas of hypertrophic gastritis may be hard to differentiate from *carcinoma*. If there is any question about the lesion, surgical removal is indicated.

Postoperative gastritis is seen chiefly about the site of operation. There may be any of the first three types of gastritis present with hyperemia, swelling, hypertrophic nodules, and even erosions and hemorrhage. Symptoms may be lacking, or varying degrees of abdominal discomfort may be present. Treatment is similar to that of hypertrophic gastritis.

Peptic Ulcer. Peptic ulcer is the result of localized solution of the mucous membrane and its underlying structures in the stomach and upper duodenum. It may be acute or chronic and may persist for years. Ulcer is more common in the male than in the female, and may occur at any age although it is most fre-

quent between twenty and fifty years. The incidence in elderly people is increasing. Kiefer and McKell² reported 152 cases of ulcer in people over sixty-five years and found 50 per cent of these had their first symptoms after sixty years. Thirty-three of their 112 cases of duodenal ulcer had obstruction, indicating a rather advanced lesion. Characteristic history was obtained in 45 per cent of these patients. This failure to have clear-cut symptoms may be in part the reason that only ten out of ninety-two cases of peptic ulcer found in 4000 autopsies on patients over sixty reported by Boles and Dunbar³ were diagnosed. A thorough history and painstaking analysis of symptoms are extremely important in any abdominal complaint especially in aged people.

The cause of ulcer is not known. It is possible that a number of factors enter into its formation. Reduction in vitality of the mucous membrane makes the tissue susceptible to the powerful digestive and corrosive action of the gastric juice and a defect results. Fatigue, trauma, spasm or embolism of arteries, infection, irregular eating and sleeping habits, improper food, an unstable nervous system, allergy, and even endocrine disturbances apparently play a part in its production and chronicity. Experimental acute ulcers heal rapidly but chronic ulcers can only be produced under exceedingly abnormal conditions.

Site. Gastric and duodenal ulcers are found only in regions subjected to the digestive action of acid gastric juice. Most lesions are, therefore, found

from the size of a pin head to ulcerated surfaces involving most of the organ. The mucosa and submucosa are always involved, the lesion often extends through the muscular coat and may perforate the peritoneal cavity or penetrate adjacent organs. Stenosis of the pyloric outlet due to swelling and scar tissue is one of the complications of ulcer in that locality. Healing of a gastric ulcer may produce local contraction and deformity.

Symptoms. The symptoms of either gastric or duodenal ulcer vary in their intensity and are essentially the same for either type.

The most important subjective symptom is that of a characteristic type of *epigastric distress*. Careful questioning reveals a definite relationship between the discomfort and the eating of meals. In the morning, before breakfast, while eating, and immediately thereafter no distress is noted and if symptoms are mild, there is usually no discomfort in the forenoon. If pain appears it often begins between nine and eleven o'clock, reaches a peak and disappears before the noon meal. The discomfort is frequently described as a gnawing, burning pressure, an empty or hungry feeling, and is more or less continuous. Between one and three hours after the noon meal it may appear again, reaches its maximum intensity, and then gradually becomes less severe and disappears before supper. If retention of the noon meal occurs, pain may continue until eating supper when the distress is relieved. One to three hours after the eve-

ning discomfort is severe there is
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of food and secretions and the pa-
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 on

food eaten during the day, largely fat and carbohydrate debris, is found in the vomitus. Relief may also be obtained by taking an antacid.

Clinical manifestations tend to occur periodically and many patients give a history of recurrent attacks over a period of many years. Symptoms are most frequently observed in spring and autumn.

The mechanism by which distress is produced is not clear. The usual mechanical and chemical irritants which cause peripheral pain do not produce visceral pain. In the absence of perigastritis or perforation, pain attributable to peptic ulcer is unusual when the stomach is empty. During an attack of pain it has been shown that when the gastric content is neutralized with an antacid, pain is relieved, also that the pain can be reproduced when the contents are again acidified with hydrochloric acid. Spasm, as a result of the irritation of the raw area, may cause some of the pain. It is probable that granulations cover the base of an ulcer soon after appropriate treatment has been started and thus, although a large defect may be visible by x ray, no pain is present.

Pain may be referred to various regions of the trunk but in the majority of patients it is confined to the epigastrium and limited to a small circumscribed area. Unless extreme, it rarely extends across the epigastrium and is only occasionally referred to the back. There may be tenderness to pressure and some patients obtain a measure of relief by making steady pressure at the level of distress. Vomiting is seldom a prominent feature yet nausea may occur as the distress increases or as a result of overfilling when pyloric obstruction is present.

Vomiting of blood is not usual and, although it can be the result of other disease, it is wise to assume ulcer until proved otherwise. Severe or continued bleeding can occur without hematemesis. Loss of only 100 cc. of blood will produce a tarry stool and continued oozing gives a positive occult blood test in the stool.

With a sudden severe hemorrhage the patient usually faints and the pulse becomes rapid and thready. Extreme pallor, thirst, and restlessness often develop. Air hunger may become pronounced. The blood pressure drops. Recurrent bleeding may take place in a day or two either as a result of dislodgment of the thrombus or of a fresh erosion into a blood vessel. Prolonged bleeding seems more prone to occur from arteriosclerotic vessels, and thus is to be watched for in elderly patients.

A marked secondary anemia and pallor are not unusual after long continued loss of blood. The appetite is usually good but, because of fear of distress after eating, the patient eats less than normally.

Gastric analysis almost always reveals the presence of hydrochloric acid, frequently in high concentration. The presence of blood is not diagnostic of ulcer, since it may have been caused by trauma. Gastric contents may not contain blood if the ulcer is in the duodenum. Microscopic analysis will reveal sarcinae or budding yeast cells when food is retained for a few days.

Visible blood is not present in the stool except when recent hemorrhage has taken place. The benzidine test for occult blood after the patient has been on a meat free diet for a few days is the most reliable index as to the amount of bleeding which is taking place.

There is nothing distinctive about the blood findings. In severe hemorrhage there is no immediate change but in a few days a moderate leukocytosis and a marked reduction in hemoglobin and red cells are evident. With repeated hemorrhage and continuous oozing a persistent secondary anemia results.

Diagnosis The presence of a peptic ulcer can usually be discovered by a carefully taken history and physical examination. Laboratory studies of the blood, urine, gastric contents, and stools as well as x rays are necessary. If these are not conclusive, the distress may be tested by the use of antacids or a trial of ulcer management. Not infrequently discomfort associated with other disorders must be eliminated.

X-ray evidence is of great value but requires considerable skill in its interpretation. Fluoroscopic visualization aided by films is invaluable. These findings must be supported by clinical and laboratory evidence. An ulcer defect

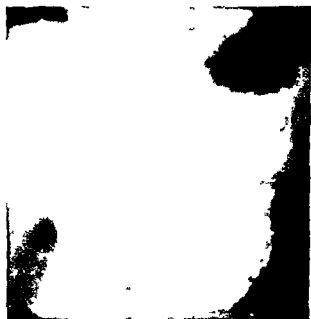


Fig. 110 Large gastric ulcer in the middle third of the lesser curvature

without spasm, irritability, or other signs of activity may be a healed lesion. Gastroscopy is of particular help in visualizing gastric ulcer, especially if there is a question as to activity or malignant change.

Complications *Pyloric obstruction* is the most common complication found. If present for a long time, the gastric muscles undergo hypertrophy and powerful peristalsis may be seen under the abdominal wall. Various degrees of retention are present, as demonstrated by the yeasty odor of the vomitus or the aspiration of food eaten many hours previously. Roentgenologically, powerful peristalses and only a fine stream of barium are seen passing through the duodenal cap.

Continued secretion or hypersecretion often accompanies pyloric obstruction. Large amounts of this juice may form each day and must be removed by aspiration at bedtime and often during the night to bring relief and promote healing.

Perigastritis and *periduodenitis* are suggested by a slight rise in temperature, an otherwise unexplained rise in leukocytes, and unusual local tenderness. Rarely, a mass develops.

Perigastric or periduodenal *adhesions* seldom produce symptoms but are not uncommonly seen at operation or autopsy.

Hour-glass stomach may be present when ordinary symptoms of ulcer are accompanied by a sense of unusual fullness after eating only a small amount of food. X-rays usually reveal the defect.

Acute perforation is one of the most serious complications of peptic ulcer. There is usually an ulcer history preceding the sudden onset of severe epigastric pain which soon becomes agonizing. It is localized at first and gradually becomes more diffuse and severe. The abdomen is retracted and assumes a board-like rigidity. The face becomes drawn, pale, and beads of cold perspiration appear on the forehead. Vomiting may occur. The temperature may be subnormal and the pulse during the first hours unchanged. The blood count may be normal. X-ray frequently shows air between the diaphragm and the liver.

Surgical intervention should be undertaken promptly since the mortality increases with each hour of delay. The severity of symptoms and degree of peritonitis are likely to be less if the stomach is nearly empty. If an operation is not undertaken a localized abscess may form, more frequently a generalized peritonitis sets in and death ensues in two to four days.

Occasionally a diagnosis of *formes frustes perforation* is made and medical management is adequate.

Malignant Degeneration The literature is replete with discussions regarding malignant degeneration of gastric ulcer. While this is possible, it is also well known that duodenal ulcer is more common than gastric ulcer, yet carcinoma of the duodenum is rare. Resistance of neoplastic cells to digestion is less than that of normal gastric cells and thus almost all gastric carcinomata are ulcerated. The first evidence of a malignancy may be the ulcer-like symptoms produced by the erosion. With this in mind all ulcers suspected of being

Some forms of cancer develop slowly but it is unlikely that a history of many years of "indigestion" means the tumor was present for that time or that the patient necessarily had a gastric ulcer during that period.

Treatment Before attempting to outline a course of treatment, all the factors relative to the lesion should be considered. Most cases will respond satisfactorily to medical management, yet surgery is imperative under certain conditions. Some indications for *surgical treatment* are:

- 1 Perforation into the abdominal cavity
- 2 Pyloric obstruction due to narrowing that fails to yield to medical measures. Advanced age or other infirmities may make surgery hazardous, in such instances bedtime aspiration, except for its inconvenience, is preferable.
- 3 Perigastric or periduodenal abscess
- 4 Gastric ulcer when recurrent or if there is reason to suspect malignancy
- 5 Deformity from ulceration, as in hour-glass stomach, to allow a better gastric function

6 Repeated massive hemorrhage

7 Removal of foci of infection

8 Gastrojejunal ulcer which fails to respond satisfactorily to medical management

The type of operation advisable depends upon the individual situation. Gastroenterostomy often provides adequate emptying and healing of a duodenal ulcer. In some instances resection may be preferable especially when hemorrhage has been a complication. More recently in some clinics, vagotomy has been combined with these operations as a further aid to promote healing and prevent recurrence.

MEDICAL TREATMENT Almost all medical treatment for over one hundred years has employed some means of reducing the digestive and corrosive action of gastric juice. Albuminous soft foods and antacids between feedings are used in various ways to control pain and facilitate healing. Gastric and duodenal ulcers should heal as readily as ulcers elsewhere in the body except for the fact that they are continuously subjected to the digestive action of gastric juice. Sippy reasoned that if gastric juice retarded the healing of an ulcer, the rational medical treatment should be directed toward lessening or completely inhibiting the disintegrating and digestive properties of the gastric juice. His treatment is directed toward the continuous neutralization of free hydrochloric acid so that not only its corrosive action is reduced but also that the hydrogen ion concentration is decreased to the point where pepsin is practically inert. This is accomplished by giving small feedings every hour and antacid on the half hour throughout the day. In this manner the nutrition is maintained, and the acidity is constantly modified. Pain due to action of gastric juice usually disappears during the first day of treatment.

A program such as this requires patience but can easily be followed in all walks of life if desired. Attempts at simplification have led to "modified Sippy managements," none of which fulfill the original requirements unless constant acid reduction is maintained. Studies with a gastric electrode placed in the antrum of the stomach have shown how difficult it is to obtain adequate neutralization.⁴

CHOICE OF FOOD Nutrition must be maintained in spite of the defect in the digestive tract. This can be done easily and effectively since foods can be utilized in maintaining reduced gastric acidity. If a vitamin deficiency should occur in spite of orange juice, milk, cream, and egg feedings, concentrates can be given by mouth or parenterally. Milk constitutes one of the main sources of nutrition and can be fortified by using a half milk and half cream mixture. Milk alone accomplishes more neutralization than a half milk and half cream mixture and is preferred because the fat in the cream tends to separate from the milk and become concentrated in the stomach, especially if there is poor emptying. Fat also reduces gastric peristalsis and has little neutralizing capacity.

At first only the soft, more effective neutralizing foods are given in small portions containing only two or three items in each feeding. As healing progresses the amount and number of foods are gradually increased. All coarse, fibrous, and highly seasoned food, beer, wine, and liquor must be avoided permanently or at least for many months after all evidence of ulceration has passed. The following diet has in our experience proven satisfactory.

A mixture of $1\frac{1}{2}$ ounces milk and $1\frac{1}{2}$ ounces cream every hour from 8:00 A.M. until 7:00 P.M. will provide

18 ounces of milk and 18 ounces of cream

Calories	1495	Phos	9 gm	Thiamine	0.38 mg
Protein	35 gm	Iron	0.7 mg	Riboflavin	1.68 mg
Calcium	1.16 gm	Vitamin A	5300 I U	Niacin	1.08 mg
				Vitamin C	10 mg

DIET TO INCLUDE THE FOLLOWING

<i>Cereals</i>	Farina, oatmeal, fine whole grain cereals, cold cereal, without bran
<i>Breadstuffs</i>	White bread, fine whole wheat bread, rye bread without seeds
<i>Beverage</i>	Milk, tea
<i>Vegetables</i>	Potatoes Baked, mashed, buttered or creamed Purée of spinach, carrot, asparagus, squash, string beans, peas
<i>Fruits</i>	Canned pears, peaches, apricots, applesauce Stewed dried prunes, apricots, peaches, rice, bananas, orange juice and grapefruit juice
<i>Desserts</i>	Simple desserts as blanc mange, custards, and puddings, gelatins, ice cream, simple cakes and cookies
<i>Soups</i>	Cream soups Chicken and beef broth, not highly seasoned
<i>Meat or Meat Substitutes</i>	Fowl, fish and bacon, roast or broiled beef and lamb
<i>Cheese</i>	Of all kinds

SAMPLE MENU

8 A.M. <i>Breakfast</i>	12 Noon <i>Dinner</i>	6 P.M. <i>Supper</i>
Orange juice	Cream soup	Poached egg on toast
Farina	Sliced chicken	Baked potato
Egg	Mashed potatoes	Mashed squash
Bacon, 1 strip	Purée carrots	Canned peaches
Toast	Ice cream	Bread, butter
Tea	Bread, butter	Tea
	Tea	

Yields approximately the following Value

Calories	1450	Vitamin A	20,000 I U
Protein	60 gm	Thiamine	0.86 mg
Calcium	0.05 gm	Riboflavin	1.33 mg
Phosphorus	1.0 gm	Niacin	8.8 mg
Iron	9 mg	Vitamin C	105 mg

CHOICE OF ANTACID A great number of neutralizing substances are available today but no ideal substance has yet been found. This ideal neutralizing agent should not disturb the acid-base balance of the body nor other normal physiologic processes, yet it must have an effective antacid property. Milk is one of the oldest and closest to the ideal that we have, yet its sole use in the amounts required is impractical. In the usual 3 ounce amounts it is, therefore, alternated with another antacid.

Calcium carbonate and sodium bicarbonate are cheap and effective alkalies but their solubility tends to upset the body chemistry, especially in the presence of renal damage and certain other conditions.

popular and have stimulated the search for better compounds. As a result, colloids, organic and inorganic, with their adsorptive properties have been explored.

After analyzing the properties of various commonly used antacids we have come to the conclusion that the tribasic forms of calcium and magnesium phosphates are our most practical aids in reducing gastric acidity. These substances are relatively insoluble, unflavored, well tolerated, inexpensive, and rarely affect the acid-base balance of the body. Tribasic calcium phosphate is constipating and the magnesium form is laxative, therefore, they are combined into one powder both to supply the amount of neutralization desired and to permit normal bowel function. As a rule a powder containing 20 grains of the calcium salt and 15 grains of the magnesium salt is satisfactory, but in some instances the proportion of one to the other must be modified or the neutralizing capacity of the entire powder increased or decreased to meet the individual needs.

TABLE 12*

GASTRIC CONTENT pH AS FOUND WHEN DIFFERENT ANTACIDS WERE USED. THE TOTAL NEUTRALIZING CAPACITY OF THE ANTACIDS USED IN EACH TEST IS SHOWN

Gastric pH Values

Test	Patient		Antacid [†] capacity
	N L B	A H	
Fasting State	1.60	1.44	0
Tribasic Calcium Phosphate 20 gr (1.3 gm)	2.04	4.02	191.8 cc
Tribasic Magnesium Phosphate 15 gr (1.0 gm)			
Calcium Carbonate 15 gr (1.0 gm)	1.70	3.28	191.6 cc
Sodium Bicarbonate 20 gr (1.3 gm)	2.27	1.92	172.6 cc
Aluminum Hydroxide (Amphojel) 10 cc	1.81	1.88	139.0 cc
Mucin (Armour & Co) 10 gm	1.33	1.88	188.2 cc
Magnesium Trisilicate (Trisomin) 10 gr (0.65 gm)	1.45	.37	139.4 cc
Milk 3 oz (90 cc)	2.40	2.90	158.8 cc

(Average pH values of gastric contents of patients N L B and A H obtained when readings were taken every 5 minutes over two hour test periods. Except in the fasting state, the test substance was given every hour and 3 oz (90 cc) of milk on the half hour.)

* Breuhäus, H C, and Eyerly, J B. Antacids. Their Effect by Titration and within the Human Stomach, *Ann Int Med*, 14

† Amount of 0.17 N HCl required to titrate amount of antacid used during test period to pH 3.5

Other means of modifying gastric acidity may be useful. Mild sedatives, such as bromides and barbiturates, aid in reducing nervous tension but must be given with caution. Atropine and belladonna can also be given but the general reaction to the doses by which practical reduction in secretion is obtained limits their usefulness. Hormones, such as uro- and enterogastrone, show promise. Diminution of secretion by x ray treatment over the gastric region is temporary and of questionable value.

TREATMENT OF UNCOMPLICATED ULCER As soon as the diagnosis of duodenal ulcer has been made, treatment should be started. When possible it is best to hospitalize the patient. The stomach contents are aspirated, usually at 4.30 P.M., and tested to determine the degree of neutralization accomplished. The individual's bowel function on the powder regimen can also be ascertained. One or two aspirations at bedtime show how the stomach is emptying.

TABLE 13

NEUTRALIZING CAPACITY OF EQUIVALENT AMOUNTS OF MILK AND ITS HYDROLYSATES

	cc N/10 HCl
57 cc regular milk (contains 2 gm protein)	43
Sofkurd fat-free powder	18
Regular skim-milk powder	16
Klim and regular sofokurd powder	12
Lactamin	34
Protein Hydrolysate, Squibb	30
Decamin	28
Protein Hydrolysate, M.R.T. no. 1	27
Aminonut	27
Amigen	26
Protolysate	24
Pendarvon	23
Boramin	23
Protenum	19
Protolac	18
Ledinac	14
Aminoid	14
Lonolac	9
Essenamine	7

2.0 gm quantities of milk products and protein hydrolysates dissolved in 150 cc distilled water and titrated with N/10 hydrochloric acid to pH 3.5

Three small feedings, consisting of one to three of the items listed in the diet, are usually allowed at the regular meal time on the following schedule

8 00 A.M.	Breakfast	3 00 P.M.	Powder
9 00 A.M.	Powder	3 30 P.M.	Milk
9 30 A.M.	Milk	4 00 P.M.	Powder
10 00 A.M.	Powder	4 30 P.M.	Milk
10 30 A.M.	Milk	5 00 P.M.	Powder
11 00 A.M.	Powder	5 30 P.M.	Milk
11 30 A.M.	Milk	6 00 P.M.	Supper
12 00 Noon	Dinner	7 00 P.M.	Milk
1 00 P.M.	Powder	7 30 P.M.	Powder
1 30 P.M.	Milk	8 00 P.M.	Powder
2 00 P.M.	Powder	8 30 P.M.	Powder
2 30 P.M.	Milk	9 00 P.M.	Powder
		9 30 P.M.	Powder or aspiration

The progress is observed at frequent intervals with the x-ray, stool analyses for occult blood, and for other evidences of healing. If signs of ulcer activity have disappeared, the program is modified after a few months, the diet is left essentially unchanged, powders are continued every hour, but a glass of milk is taken in midmorning and at two and four o'clock in the afternoon, the evening powders are taken hourly. Gradually all powder and milk may be omitted and a normal diet resumed.

TREATMENT WHEN PYLORIC OBSTRUCTION IS PRESENT Pyloric obstruction is more frequent in older individuals because of previous ulceration with residual scarring. Spasm and edema will also cause gastric retention. In some instances the outlet may be markedly constricted but because of powerful compensatory gastric hyperperistalsis little retention occurs. All patients with this complication should be given bed rest during the acute phase of obstruction. A half hour schedule without food other than milk is given for the first few days. Warm moist blanket packs to the abdomen during the day aid in reducing spasm. The stomach is emptied, and often lavaged with one or two bulbfuls of warm water at 9:30 P. M. every night until only 1 to 3 ounces are obtained. If there is night pain or other evidence of continued secretion, a small tube may be left in the stomach and this secretion withdrawn periodically during the night. Constriction due to inflammation and edema will subside after several days and aspirations during the night may be stopped but the bedtime aspirations should be continued, sometimes for months, until retention is relieved. Chloride deficit may occur if large amounts of hydrochloric acid are removed by aspiration, this should be watched so that such loss can be counteracted by intravenous infusions of salt solution. Dehydration must be treated in the usual manner.

Most cases of pyloric obstruction will be relieved in a few weeks or months if the above treatment is carried out. If in spite of these measures scar tissue contraction does not permit adequate emptying surgery is advisable. Since recurrence is not uncommon and healing is slower when chronic changes are present, the period of observation must be longer and changes in management must be made more slowly than when no complications are present.

When periduodenitis or perigastritis is present, rest in bed and heat to the abdomen plus the usual therapy are sufficient. An abscess may resolve or have to be drained surgically.

Gastric ulcers may penetrate other organs or retard gastric peristalsis as a result of adhesions, scar formation, or spasm, to produce mechanical dysfunction.

when no marked retention is present

TREATMENT OF MASSIVE HEMORRHAGE All ulcers bleed to some extent as a result of erosion of capillaries but when a large vessel is penetrated the amount of blood lost will depend upon the caliber of that vessel. Death may result from one hemorrhage or from several copious hemorrhages over a period of a few hours or days. Surgical intervention is tempting but the operation is very difficult and is particularly hazardous in such an emergency. Results from careful medical management have proved most satisfactory in our hands. Immediate absolute bed rest is instituted. Restlessness is controlled with pan-
topon or barbiturates hypodermically. Blood is drawn and typed so that transfusions can be given at any time. Nothing is given by mouth for twenty-four to forty-eight hours, ice chips may be sucked to overcome thirst. The pulse is taken frequently but the patient is not otherwise disturbed. If the pulse be-
comes rapid and the blood is voided in
stool or retard clot
formation or loosen the thrombus. Usually the half hour schedule is begun

after thirty six to forty-eight hours if there is no evidence of further bleeding. Food other than milk is withheld for several days after hemorrhage. Absolute bed rest is maintained for at least two weeks, after which time the patient may be gently fluoroscoped to get a better impression as to the nature of the lesion.

In a few instances nausea and vomiting, due to accumulation of blood in the stomach, will have to be relieved by gentle aspiration. An astringent antacid, such as aluminum hydroxide, may be useful in the first few days and may then be replaced by the usual powder.

Gastric Carcinoma In spite of all advances in the diagnosis and treatment of stomach disease, gastric carcinoma remains one of the most insidious forms of cancer. Alcohol, tobacco, irritating foods, gastric ulcer, gastritis, trauma, worry, etc., have been accused. Heredity may predispose to this condition. The idea of chronic irritation leading to malignant change seems plausible but is not proved in our experience. The decision as to whether inflammatory or malignant changes are present may be uncertain, even when a resected ulcer specimen is examined under the microscope.

Pathology The essential pathologic change is that produced by malignant proliferation of epithelial cells of the mucosa with secondary degenerative changes and metastases into other organs.

The cylindrical cell adenocarcinoma is the most common type.

Symptoms. Unfortunately symptoms are rarely present until the tumor has grown large enough to impair normal gastric function or produce pain due to ulceration. If neither of these is present a surprisingly large growth may develop before medical attention is sought. The onset is often mild with gradually increasing discomfort, or it may come on with sudden severity in people who have previously enjoyed a "cast iron" stomach. Pain is usually one of the first symptoms, it is often continuous, aggravated by eating, and may be described as a burning or gnawing in the epigastrium, it may be referred or radiate to the back and shoulders. Tenderness to pressure in the epigastrium is not uncommon. Food or alkali give little relief and soft foods are usually chosen.

Vomiting is a common symptom. It may occur at intervals in the early stages but as obstruction increases almost all food may be vomited. The vomitus may have a sweet odor or, if decomposition and necrosis of the tumor are present, it will be offensive. Nausea, because of distention of the stomach walls or as a result of tumor infiltration, may be present. Little food is taken. marked weight loss occurs, and constipation is the rule.

Hemorrhage is not infrequent but it is usually not so severe as in ulcer. Slight oozing from the tumor produces coffee ground vomitus and may be sufficient to make the stools dark. Occult blood is always found in the stools and gastric contents.

The appetite is poor and a particular dislike for meat occurs. Appetite may return temporarily but even after a palliative operation such patients often avoid food. Loss of strength is proportionate to the appetite loss. Secondary anemia due to the loss of blood and the toxic effect of the growth is marked and the skin takes on a characteristic lemon yellow tint. A low grade fever is usually present. Edema of the lower extremities is consistently seen in the late phase.

On physical examination almost nothing abnormal will be found in the early stages. Later on epigastric tumor mass may be palpated and peristalses seen under the abdominal wall. Metastases to the liver and skin are not un-

7



Fig 111 Carcinoma of the distal third of the stomach



Fig 112 Small carcinoma of antrum of stomach

common Occasionally a mass in the pouch of Douglas is found on rectal examination Peritoneal carcinomatosis and extension into the mesenteric

glands will produce ascites *Jaundice* will be found if the bile passages are compressed

Neuralgias may occur in various parts of the body as a result of malnutrition, pressure on sensory nerves or invasion of bone

Laboratory Findings Gastric analysis is very helpful A reduction in gastric acidity, or no free acid, is common, the amount of acid found often corresponds to the extent of the invasion of the stomach Blood, either fresh or of the "coffee ground" type, is commonly found If acid reduction is marked and obstruction present, lactic acid (Boas Oppler) bacilli are usually present Cancer cells are occasionally seen Vomitus may be profuse The odor is characteristic of necrotic tissue or decomposition of food if there is marked retention and no acid

Occult blood is found in the stools



Fig 113 Carcinoma of the lesser curvature of the stomach resembling benign ulcer

There may be no change in the blood picture in the early stages but as the condition advances the constant bleeding produces a secondary anemia which becomes severe Invasion of surrounding tissues metastases infection, and necrosis of the tumor cause a mild elevation in the white blood cell count

X ray and Gastroscopic Findings These two aids are invaluable in the diagnosis of cancer of the stomach In every instance where gastric malignancy is suspected the stomach should be observed under the fluoroscope Filling defects, changes in peristalsis and flexibility of the stomach walls can be observed Location and size of the lesion are important factors determined by x rays All gastric ulcers should be frequently observed by this means so that healing or malignancy may be determined

With the gastroscope almost all portions of the stomach can be seen Thus is an aid in determining the nature of a defect seen by x ray and in some

instances will reveal lesions before they are large enough to be detected by other methods. Frequently the difference between ulcer and carcinoma can be definitely decided by this means, occasionally, when gastric acid values are unchanged, erosion of the tumor may be so extensive that even the gastroscopist has difficulty in deciding between ulcer and cancer. As a rule the picture of raised nodular edges about a necrotic dirty ulcer base or of a fungating bleeding tumor is characteristic enough to make a diagnosis. The extent of invasion of the adjacent stomach wall can also be seen and the practicability of surgery determined.

Diagnosis Until some method of detecting cancer early is made available it is unlikely that deaths from cancer of the stomach will be reduced materially. Its onset is often so casual that in most instances medical attention is sought only after symptoms of obstruction or invasion of other structures are present. Greater alertness on the part of the patient and physician to the possibilities of malignancy in people past middle age who have attacks of stomach trouble will help discover more early lesions. Symptomatic treatment without consideration of the type of discomfort, and without stool analyses, gastric analyses, x rays and gastroscopy favor the loss of time in which early lesions can be found.

A history of good health prior to the appearance of discomfort or a change from ordinary abdominal sensations, with loss of weight and anemia, should be thoroughly investigated. If there is a question about gastric ulcer or other findings are inconclusive, a gastroscopy should be done. Trial medical management of a gastric ulcer of questionable nature may be attempted but if relief of symptoms and obvious healing do not occur promptly it is safest to resect the lesion.

Treatment As in other forms of cancer, the only hope of cure lies in early resection. Considerable difference in opinion exists as to the advisability of surgery in some instances. In large tumors, *metastases* are almost certain while in small lesions they are frequently present. As a rule, removal of small tumors gives greatest satisfaction, although only a small percentage outlive a five year period or are cured. X-rays and gastroscopy are of great help to the surgeon in deciding the size and extent of invasion by the tumor. If the condition is inoperable and no obstruction is present, the patient is best put on a soft semiliquid diet. Vomiting and marked retention can frequently be relieved with a *gastroenterostomy*. Often this procedure is followed by considerable temporary improvement which not only prolongs life but adds much to the patient's comfort. Jejunostomy is seldom advisable, digestion about the stomach and the difficult feeding problem only add to an already difficult condition.

Sedatives of the bromide or barbiturate type can be used for restlessness. Salicylates may be sufficient to control early pain but sooner or later opiates are necessary. Fear of addiction to opiates is inconsequential since the patient will die from his neoplasm before addiction becomes a problem. It is best first to try a mild opiate, such as codeine, by mouth. Later stronger medication may be given by mouth and when this becomes ineffective the hypodermic route is necessary.

Benign Tumors. These tumors are rare and consist chiefly of polyp-like growths. Among them are papillomata, myomata, fibromata, and lymphadenomata. If pedunculated they may produce a ball-valve effect in the pylorus and cause intermittent obstruction and hemorrhage. Since malignant degeneration must always be feared, they are best removed surgically.

Sarcoma. This tumor arises from the muscular or fibrous portion of the stomach wall below the gastric mucosa and ulcerates late. It is rare and produces symptoms like carcinoma. The diagnosis is occasionally made by finding the tumor cells in gastric washings. Early resection is advisable if possible, but the prognosis is usually hopeless. General treatment is the same as that of carcinoma.

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CHAPTER 34

DISEASES OF THE INTESTINES

FRANK C. VALDEZ

INTRODUCTION

COMPLAINTS of intestinal disorders are among the most frequent difficulties encountered in the aged. The subjective symptoms are frequently functional in origin and due largely to mental maladjustments. Inability to adjust to modern activities, a sense of neglect, economic, social, and familial insecurity are common sources of anxiety, which is reflected in the bowel. However, it is utterly unwarranted to assume that all complaints referable to the intestines are of psychogenic origin. Organic conditions, many of them dangerous, are common, and often obscured by the lack of subjective symptoms (See p. 33). A determined effort must be made to rule out organic disease, and this requires the use of all available laboratory facilities and especially a very complete clinical history and physical examination. Time and patience are necessary for thoroughness. But the results are worth the effort expended.

Abnormal conditions of the small intestine are not common. Organic lesions occurring in older people are usually slow in onset and difficult to diagnose. Every diagnostic procedure at the disposal of the clinician should be used. Examination of the feces and roentgenoscopic study must not be omitted. Symptoms referable to faulty absorption in the small intestine are vague and indefinite and frequently not directly associated with the alimentary tract. Malnutrition may arise as a result of inefficient absorption of essential elements as well as from inadequate ingestion (See Chapter 12). Rafsky and Newman¹ have shown that certain vitamins are poorly absorbed by the aged and suspect that more calories are needed for old people than is commonly believed. Jacob Meyer² states that the nutrition of the aged is dependent on factors other than the number of calories taken. According to his findings there are very few, if any, symptoms in older people referable to faulty carbohydrate digestion. Advanced age is associated with a slowly progressive atrophy of the *intestinal mucosa and musculature alike* (See Chapter 3). *Secretory function* is depressed and thus the digestive enzymes are less effective than in youth.³

VOLVULUS

Volvulus of the small intestine occurs relatively frequently in old people. The aged usually have a relaxation of the abdominal wall and less mesenteric fat. Hernia and postoperative adhesions, both more common in later years, contribute to the frequency of volvulus. The *symptoms* are primarily those of intestinal obstruction with an onset that may be gradual or abrupt. If obstruction develops slowly, there occurs a mild, intermittent pain in the middle abdomen or epigastric region, which increases in severity and frequency of occurrence as the obstruction becomes more pronounced. Abdominal disten-

tion, tenderness, and muscular rigidity follow later. Visible peristalsis is not common, but may be present. Listening to the change in the bowel sounds will

pain is severe and nausea and vomiting occur early. Marked abdominal distention, muscular tenderness, and rigidity follow more rapidly. The temperature is elevated and white blood corpuscles are moderately increased.

A preoperative *diagnosis* of volvulus is infrequent. Roentgenologic study should never be omitted, a flat plate of the abdomen reveals the gross bowel distention and assists in localizing the site of obstruction. Intestinal obstruction always warrants *surgical intervention*; the exact cause is best determined at the time of operation. The surgeon must be prepared for the possibility of extensive resection, for mesenteric thrombosis and/or pancreatitis may readily present the clinical picture of volvulus with obstruction. These patients are poor risks, but prompt surgical intervention offers the only hope and should never be denied the older patient just because he is old. Gangrene occurs more quickly in the aged, partly because the tissues are more sensitive to histanoxia and partly because vascular changes interfere still more with the impaired blood supply. Whether to resect the portion of the intestine involved in the volvulus is a question that is best answered at the operating table. Recurrences of volvulus in the bowel that is not resected are common.

Preoperative attention to nutrition, and the reestablishment of water and electrolytic balance, are essential. Vomiting frequently depletes these patients of water and chlorides. Transfusions of saline, dextrose, blood, and plasma before, during, and after operation are significantly reducing the mortality of intestinal surgery in the aged (see Chapter 8). Disturbed protein concentration in the blood and tissue fluids is perhaps as significant as asphyxia in inducing edema of the intestinal walls and thus impairing healing. The homeostatic equilibrium and nutrition of the patient as a whole demand as much attention, or more, from internist and surgeon, than do the local lesions.

APPENDICITIS

Appendicitis does not occur frequently in old people, but it is more frequent than many realize. Because the clinical picture and laboratory findings are often atypical, appendicitis in the aged is often unrecognized. Maes⁴ attributes the unusual findings to atrophy of the lymphoid tissue which predisposes to a more rapid spread of the infection, and to the "naturally lowered resistance of elderly patients." Arteriosclerotic changes probably contribute to the rapid development of gangrene and abscess formation observed in some cases. In young people, appendicitis is essentially a local infection spreading through lymphatic channels. Gangrene occurs late and is usually patchy and localized. In the aged, on the other hand, appendicitis is largely a vascular disease and massive gangrene is frequent and often early. Boyce⁵ has pointed out that these differences account for the greater seriousness of appendicitis in later years.

Diagnosis. The clinical picture of appendicitis in later years is more often distorted than typical though in many instances the symptoms and signs may be as classically typical as in youth. However, the variation in presenting complaints and clinical observations is so great that the disorder must be con-

sidered a possibility whenever a previously well older person complains of unexplained vague digestive distress

The pain may not be severe, but is usually acute in onset with a rapid localization to the right side of the abdomen. Localized tenderness is usually present, but may disappear more rapidly than in younger people. The leukocyte count is variable, often the increase is negligible despite serous and purulent infection. Fever is a most uncertain guide. Even in the presence of abscess formation and peritonitis, the febrile response may be very meager (See p 33). Muscular rigidity is not definite and may be wholly lacking. Abnormal location of the appendix is not uncommon. Retrocecal or subhepatic appendicitis may be confused with intestinal obstruction or acute cholecystitis. As pyosalpingitis is very rare in the elderly, a rectal examination is even more helpful than in younger women in identifying a retrocecal appendicitis. It should never be omitted in the examination of patients with right lower quadrant pain. If the appendix lies over the ureter, the clinical picture may suggest ureteral spasm, obstruction, or pyelitis, and blood may be found in the urine. The x-ray is not helpful in diagnosis.

Treatment. The treatment of appendicitis in the elderly is the same as in the younger patient.

drainage. As the mortality from appendicitis increases with age, almost doubling from the age group 45-54 (11.1 per 100,000) to 65-74 (19.6 per 100,000) in 1940, prompt surgical intervention is even more urgent than with younger patients. Taylor⁶ gives the following timetable as typical of appendicitis in the aged: engorgement the first day, gangrene on the second, peritonitis and multiple abscess formation on the third. Progression may be even more rapid. Peritonitis in the first twenty-four hours is not uncommon.

CONSTIPATION

Constipation is the most frequent complaint of the aged. A clear and definite understanding of what constitutes constipation must be presented to these individuals before much can be accomplished. Some believe that three watery stools must be passed daily while others are convinced that a certain amount of feces must be in the stool. It is not the frequency of defecations but the consistency of the stool that determines whether an individual is constipated. There are many who pass a normal stool every two or three days and are in perfect health with a sense of great well-being. The normal stool is soft and formed, and the amount is not of much consequence.

The more common *etiological factors* of constipation encountered in older people are: (1) diminished function in the muscles involved in defecation such as the diaphragm, abdominal muscles, and the muscles of the intestines and anus, (2) the laxative habit, (3) colonic irrigations, (4) poor dietary habits involving insufficient bulk, perhaps influenced by a lack of teeth, (5) inadequate fluid intake, and (6) failure to heed the desire to defecate.

In old age the muscles involved in the act of defecation may lose much of their tone. The cooperative action between the extrinsic and the intrinsic muscles of the large intestine is impaired. The tone of the rectum is decreased. The laxative habit is common in the elderly. It may be due to a habit of using laxatives or to a habit of using enemas.

observation All kinds of cathartics have been taken at various intervals, often for many months and years Laxatives cause the individual to pass loose or watery stools, and as a result of overemptying there is often no defecation the next day This is taken by the patient as an indication to take another cathartic The normal functioning of the intestines is completely upset Because of a lack of appetite and poor teeth the diet frequently does not contain food in which there is sufficient bulky waste material to stimulate peristalsis The habit of repeated colonic irrigations is often started by some cultist and continued in "irrigation stations" Once started, this vicious habit may be continued by the patients at home I have had numerous patients who used many gallons of water to irrigate and "empty the whole bowel" daily Most of them have been over fifty years of age Such practices completely wipe out the desire to defecate

The patient who is constipated may suffer no subjective distress whatever, or may complain of a feeling of fullness, shifting abdominal distress or pain, dull aching sensations in the lumbar region, headache, and a feeling in the head that is described as dizziness, fullness, or lightheadedness These symptoms, and many others, vary in severity with the patient, and disappear with surprising promptness after a bowel movement I have seldom seen anyone with severe pain in the abdomen referable to constipation

Accumulation of feces in the rectum may cause rectal tenseness, and a feeling of fullness or pressure Painful complications such as hemorrhoids fissures and infections of the papillae may occur

Many elderly people complain about "biliousness," which they attribute to autointoxication My experience has been like that of Alvarez,⁷ who states that the patient who is inclined to fear intestinal autointoxication can be cured by being told the truth Time spent in explaining the untruthfulness of this persistent concept, frequently expressed as "torpid liver" or "intestinal toxemia," is well rewarded Most patients do not realize that the character of the stool is immensely more significant than the frequency of defecation A hard dry stool is a constipated evacuation mushy or watery stools indicate diarrhea whether frequent or infrequent

Constipation is a symptom, not a disease Thus, diagnosis is concerned almost solely with finding the *reasons* for the poor bowel functioning Though the etiologic factors enumerated above account for the vast majority of instances of constipation, it must not be forgotten that the symptom may be indicative of serious organic disease A complete physical examination including rectal palpation, should be the invariable rule Rectal examination

habits, perhaps some weight loss, and increasing weakness

Treatment In the aged the most important part of the treatment is education of the patient in the physiology of the intestines in understandable phraseology All cathartics should be stopped I have rarely found it necessary to use any of the hydrophilic colloids, and I fail to see the advantage of lubricating the entire gastrointestinal tract with mineral oil in order to empty the contents of the rectum It should also be recognized that old people do not absorb vitamins as readily as younger people and mineral oil interferes with the absorption of the fat soluble vitamins

The *dietary management* of constipation must be individualized. It is essential that the patient understands that stools are made of waste material, and that sufficient amount of food containing waste must be eaten. Fruit may not furnish bulk, but the chemical process which it goes through in digestion stimulates peristalsis. Three meals daily are prescribed, with emphasis on a liberal breakfast. Two vegetables and a fruit should be eaten at the noon and evening meals. The vegetables must be prepared in such a way that the patient can eat them, and this depends upon his teeth. It helps to tell each individual what he should eat, and that roughly the vegetables that grow above the ground are preferable. Cooked vegetables are as a rule less irritating than raw vegetables. If they do not cause abdominal distress, a mixture of prunes, dates, and figs may be given in the stubborn cases. (See p. 132.)

A regular habit time for defecation is essential, and the best time is after breakfast. Ample time should be taken for a bowel movement, and once the habit is established, acceptance of the normal postprandial urge should not be postponed. Upon arising in the morning, two glasses of hot water should be taken, and in the stubborn cases, a glycerin suppository inserted in the rectum. As pointed out, there is a diminished tone in the muscles that aid in defecation. Exercises to increase the tone of the abdominal muscles may be prescribed, if there is not some contraindication, such as an extensive ventral hernia.

Two or three ounces of *vegetable or salad oil* (not mineral oil) introduced as a retention enema may be a valuable aid, and it is best to insert the oil at bedtime, providing there is no leaking during the night. It should be understood that this aid is to be discontinued as soon as possible. Water enemas may be resorted to if necessary. However, not more than one pint of warm water should be used at one time. Enemas should not be encouraged as a routine. Large enemas, colonic irrigations, and the use of a cascade are forbidden.

If abdominal distress is present, *tincture of belladonna* may be given before meal time for six days, stopped for three days, then repeated. A longer period on this medication may cause dryness of the mouth and blurring of the vision which is quite disagreeable and alarming to some people. Other antispasmodics, such as *trasentine*, *pavatrine*, and *syntropan*, may be used if *atropine* is poorly tolerated. Dry heat to the abdomen also gives desirable relief to the sensation of cramping. *Inductotherm* and the *infra-red lamp* may also be used in certain cases to advantage.

It should be repeated that constipation worries elderly people more than any other condition. Due to persistent wrong preconceived ideas about the assumed importance of daily bowel movements, successful treatment often requires considerable patience, but adherence to general principles will give satisfactory results. Fecal impaction, as a complication of constipation, is best treated prophylactically.

FECAL IMPACTION

Fecal impaction is frequent in the old age group. This is especially true in institutions and old people's homes, or where the senile are highly inactive or bedridden. Accumulation of feces may occur at any place in the colon, but occurs most commonly in the rectum. The fecal material is usually a formed mass, with the consistency of firm putty.

Fecal impaction is invariably a sequel of constipation, the impacted mass is an exaggeratedly constipated stool. The desire for defecation is neglected

until the rectal reflex is lost, thus allowing feces to accumulate in the anal area without causing any symptoms until the mass is large and quite firm. This occurs most often in individuals who have not trained themselves to have a regular time for bowel evacuation.

It cannot be overemphasized that medications may induce fecal impactions. Kaolin, aluminum preparations, calcium carbonate, bismuth, and hydroscopic gums encourage the formation of fecal masses. These fecal masses may extend from the rectum into the sigmoid colon. It is necessary to be careful in giving such drugs to the aged, especially in those with redundant colon, congenital megacolon, or stricture formation from any cause. Intestinal obstruction requiring surgical procedure has occurred as a result of impaction. Gallstones that have been passed may lodge in the rectum and form the nucleus of a fecal mass. Benson and Barger⁸ report such a case in a woman sixty nine years of age.



Fig. 114 Fecal impaction of the rectum

Arthritis, heart conditions, senile sclerosis with hemiparesis, general weakness, and other chronic ailments make it necessary for many old people to stay in bed most of the time. Not infrequently the diets of these individuals are of the low residue type, their bowel habits are irregular, and are frequently neglected. As has been pointed out, the rectal ampulla is often dilated to a large size in the senile, making it necessary to exercise special care or fecal impaction will occur. It should also be recognized that psychotic patients need special care to prevent a fecal impaction. A rectal examination should be made at regular intervals in such patients.

Barium used in roentgenologic examination may cause fecal impaction at any age, but special care must be exercised to prevent it in old people. I have found it of great benefit to make a rectal examination of all patients twenty-four hours after a barium meal or a barium enema. A barium impaction is difficult to remove, and occasionally damage is done to the rectum in doing

so If there is any suspicion of an impaction above the rectum, a fluoroscopic examination should be made

Those in the old age group who undergo surgical operations must be watched carefully if a fecal impaction is to be avoided It should be more generally recognized that a rectal examination before any operation is advisable Narcotics given to relieve pain encourage constipation and thus the formation of a fecal mass in the rectum Several cases of short circuit operations for colonic malignancy have come under my observation following the operation because of fecal impaction (Fig 114)

Diseases of the rectum may be the etiologic explanation of fecal impactions

Symptoms The subjective symptoms of fecal impaction are numerous and similar to those associated with constipation Symptoms referable to the rectum are common and often distressing A frequent objective symptom is small watery evacuations that pass around the fecal mass It is possible for a large mass to become cannulated, allowing watery material to pass through The mass may also be hard enough to break the mucous membrane and cause bleeding Ulceration with bleeding can occur Pressure of the hard fecal mass against the bladder may cause urinary symptoms and even incontinence Uremic findings in a man sixty seven due to a fecal mass the size of a cricket ball have been reported by Dodd⁹ Rarely is the fecal mass hard enough to cause a perforation of the bowel with the symptoms and findings of peritonitis or abscess formation

Diagnosis The diagnosis of fecal impaction is very simple because it usually occurs in the rectum, and a digital examination of the rectum is all that is necessary If the impaction is in the sigmoid region, sigmoidoscopic examination will reveal its presence

Impactions occurring in other portions of the colon may have to be differentiated from tumors A fecal mass is usually more freely movable, and likely to be long and sausage shaped More of the colon may be involved than is probable with a tumor Clinical findings in cases of impaction do not reveal the loss of weight, severe anemia, increased sedimentation rate, or the typical slowly developing weakness characteristic of neoplastic disease Loss of appetite is not a dependable finding, as many old people have poor appetites Roentgenographic examination will be of value if the mass is in the colon

Treatment. The management to be followed when a fecal impaction is found varies with the size and consistency of the mass Small masses may be broken up and removed manually, but it is advisable to break the larger masses up, and follow through with the instillation of 3 or 4 ounces of warm vegetable oil The oil retention enema should be retained for several hours It is usually necessary to repeat this procedure several times before the mass is completely removed If not too irritating, 1 or 2 ounces of glycerin may be added to the oil If this procedure fails, small water enemas should be used It should be understood that time and gentleness are absolute necessities Hydrogen peroxide is undesirable as it causes great rectal irritation Occasionally it is necessary to anesthetize the individual in order to completely empty the rectum of a very hard mass of feces When the impaction is in the colon, mineral oil by mouth often softens the mass sufficiently to permit spontaneous passage Very small doses of magnesium salts may be used, but catharsis of any kind is a hazardous procedure Surgical intervention may become necessary when neg

lected impactions cause intestinal obstruction. After a rectal impaction is removed, special care should be taken to prevent recurrence.

INTESTINAL GAS

found. Abnormal carbohydrate fermentation may be a dominating factor in many individuals.

The *etiology* of carbohydrate dyspepsia has not been definitely determined. *Clostridium welchii* is found in the stools in most of the cases. For many years this was considered of diagnostic significance, but more recently it has been shown that this organism is also found in the stools of many individuals. Excessive peristalsis of the small intestine, deficient pancreatic secretion, a diminution in the amount of gas absorbed, and the excessive use of starches are other causes.

The *clinical picture* is characterized by an intermittent, shifting tenderness of the abdomen that may be painful, and is accompanied by a feeling of tension. Rumbling and gurgling in the intestines are very pronounced. Flatulence is a very bothersome symptom. The stools are usually loose at first when the patient consults a physician. Their passage may be quite easy, but the loose stools continue for a short time, and are followed by a period when the bowel movements are hard and formed. The distress is aggravated on eating and arises while eating, or very soon afterwards. Past history may show a period of the symptoms of an irritable colon syndrome.

The stools are gassy, acid in reaction, and *Clostridium welchii* is often found. Starch granules are easily demonstrated. Fermentation is usually not by some to rule out putrefaction. When due to putrefaction, the stools are entirely alkaline and the odor of the gas is foul. X-ray examination usually reveals organic lesions. Sigmoidoscopic examination is not helpful. It is considered unnecessary unless the x-ray visualization of the colon reveals suspicious abnormalities. There is no characteristic blood in the stools, but there may be a microcytic anemia. It should be understood that the stools are soft and formed, but a characteristic of the disorder is the excessive gas and the of the bowel movements.

Treatment. The treatment of carbohydrate dyspepsia, when it is due to

per cent fruits and vegetables are permitted. If the stools are hard, kaolin, calcium carbonate with calcium phosphate may be given three or four times daily. Antispasmodics, such as atropine, scopolamine, and pavlatine with small doses of morphine are helpful in reducing spasticity of the intestines. Hydration is an invaluable aid.

If the gas is caused by putrefaction, protein foods, such as meat, fish, and eggs, kinds, peas, nuts, and sweet potatoes are prohibited.

these are only a few of the causes of what the patient calls "gas on his stomach" with distention. Belching is an annoying symptom and will be most frequently found to be caused by swallowing air. It will require much time and patience to manage some of the cases, but if a diligent effort is made, the prognosis is good.

PANCREATITIS

Acute pancreatitis with its sudden onset of violent pain, is one of the most alarming conditions that occurs in the abdomen. Pancreatic necrosis with hemorrhage has been considered the typical pathologic change, but recently an acute pancreatitis associated with edema has been described.¹⁰ Archibald¹¹ does not accept obstruction of the pancreatic duct as an *etiological factor* and thinks that retrojection of bile or duodenal contents containing bile furnishes the best explanation for acute pancreatitis. Other causes, stated briefly, are (1) infections that may spread from adjoining tissue or through the blood or lymph stream, (2) trauma and (3) some vascular accident.

The *onset* is acute with an excruciating pain located in the mid-epigastrium or slightly to the right or left. In my experience most of the pain has occurred to the left of the epigastrium. Radiation of the pain to the lumbar region is common. Vomiting accompanies the pain. Elman has observed that prostration without shock is common in acute pancreatitis due to edema.¹⁰ In pancreatic necrosis shock is an important part of the clinical picture. The pulse is not rapid and fever is usually moderate, though the temperature may rise to 103° F. There is a tenderness in the mid epigastrium with no board like rigidity. The lips are cyanotic. Davis¹² has described an unusual case of pancreatic necrosis with cyanotic and brownish spots on the body. In acute pancreatitis due to edema there is usually a history of previous attacks. Elman¹³ has emphasized that the nonhemorrhagic form of pancreatitis associated with edema may subside spontaneously. Pancreatic necrosis is almost invariably fatal.

Laboratory findings are diagnostically significant. The leukocyte count is moderately increased, there is an elevated icteric index, and, most important there is a definite increase in the blood amylase content.¹⁴ The blood amylase decreases in pancreatitis due to edema. The blood lipase is also increased. Edmondson and Buse¹⁵ found a low blood calcium in pancreatic necrosis and thought that when the blood calcium was below 9 mg. per 100 cc. of blood the prognosis was poor. If the determination was below 7 mg. the outcome was fatal. Elman found glycosuria in six out of thirty seven cases. There is usually a mild hyperglycemia.

leukocyte count rise sharply and a board like muscular rigidity in the epigastrium is demonstrable. A survey x ray film of the abdomen may reveal an air bubble below the diaphragm or rarely in the lower right abdomen. I have seen the large air bubble in the left lower abdomen. The patient is usually d in the scapula indicus the ap-

pendix may point toward the liver and make the differentiation more difficult. *Kidney colic* causes a tenderness in the costovertebral angle, radiation of pain is to the groin, and blood, pus, or both are usually found in the urine.

Treatment. The treatment of pancreatic necrosis is invariably surgical. The surgical procedure to be followed depends upon the complications found and the technical ability of the surgeon. Though an emergency, preoperative preparation must not be neglected (see Chapter 8). Acute pancreatitis due to edema may be treated medically, by conservative symptomatic measures with small meals, and perhaps temporary parenteral feeding.

CHRONIC PANCREATITIS

Chronic pancreatitis is slow in onset and is usually secondary to cholelith disease or duodenal pathology. Long continued alcoholism and cirrhosis of the liver may also be etiologic factors. Comfort, Gambill, and Baggenstoss²² observed twenty-nine cases of chronic relapsing pancreatitis without associated disease of the biliary or gastrointestinal tract.

The head of the pancreas is the most frequent site of involvement. Jaundice increases slowly and there is no definite localized pain. Muscular tenderness and rigidity are not remarkable. The icteric index is increased, and there may be no bile in the stool. Blood in the stool may be important in differentiating from carcinoma of the head of the pancreas. This differentiation may be difficult, even when the abdomen is opened and the lesion can be palpated.

The treatment is surgical. Because of the malnourishment which is part of the disease (failure of biliary and pancreatic secretions to reach the bowel) particular attention must be paid to the pre- and postoperative nutrition of the patient. (See Chapter 8.)

COLONIC DIVERTICULITIS

Diverticula of the colon occur predominately in those of advanced age. Most of the cases are found in the sigmoid colon.

Individuals were over fifty years of age. Thus far no definite or universal cause for diverticula has been found. However, several theories have been advanced. Spriggs and Marxer¹⁷ thought that local infection was a probable explanation. Some clinicians attribute the diverticula to straining at stool, while many others believe that a weakness of the intestinal wall around the blood vessels provides the best explanation.

It is questionable as to whether diverticula, in the absence of inflammation, cause symptoms. If no other cause can be found for abdominal discomfort, it seems reasonable to me that diverticula can cause enough irritation of the bowel to initiate abnormal spasms, which in turn are capable of causing distress or pain. Diverticulitis very definitely does cause abdominal pain. Ochsner and Bagen¹⁸ reported that 89 per cent of a series of cases they observed had pain. A feeling of fullness, a generalized abdominal distress, cramp-like in type, and acute pain in the left side of the abdomen are the most frequent complaints. In my observation, pain in the left lower abdomen is quite common in diverticulitis and uncommon in diverticulosis. Pain in the right side of the abdomen is comparatively rare, even though diverticula can be

demonstrated in that portion of the bowel. There is, as a rule, no relationship to food taking, but if a relationship is noted, the pain occurs very soon after eating.

The temperature is usually below 100° F, unless complications are present. Leukocytosis is never extreme, but slight elevations of the white cell count are usual. Anemia is rare. If red meats and other sources of blood are omitted from the diet, occult blood is often found in the stools. Five to 7 per cent of seventy-two cases reported by Willard and Bockus¹⁹ showed evidence of intestinal bleeding, and in one instance gross hemorrhage occurred. Valdez, Gilbert, and Kash¹⁶ found gross blood in eighteen cases (7.6 per cent) and occult blood in thirty cases (12.8 per cent) in a series of 234 instances of diverticulitis. It should be understood that gross hemorrhage does occur, but not often.



Fig. 115. Diverticulitis that caused a massive bowel hemorrhage.

Intestinal obstruction and perforation, with or without abscess formation, are the most common complications of colonic diverticulitis. Fistula formation is not frequent. The notion that carcinoma is a common complication persists in spite of evidence to the contrary. Rankin and Brown²⁰ found carcinoma as a complication very rarely, in 227 cases of known diverticulosis only four had a coincident malignant tumor, and in 679 cases of carcinoma of the bowel only four cases of diverticulosis were found. However, discovery of diverticula does not rule out the possibility of carcinoma. There may be carcinoma in one portion of the colon and diverticulitis in another part. (See Fig. 118.)

Roentgenologic examination is essential for a diagnosis of colonic diverticula. There is no uniformity of opinion as to the roentgenoscopic diagnosis of diverticulitis. Willard and Bockus¹⁹ call attention to an angulation in the colon at the site of the diverticulitis. It seems reasonable to believe that a marked localized spasm in the region of the diverticula, not present else

where in the colon, is sufficient evidence for a roentgenoscopic diagnosis of diverticulitis.

Treatment. The treatment may be medical or surgical. Such complications as acute perforation, fistula, abscess, and coincident malignancy must be treated surgically. Partial obstruction in the sigmoid region occurs frequently. After the findings are evaluated, surgery may or may not be the procedure of choice. Sometimes abscess formation and mild obstructions can be treated successfully by medical methods. This is especially true since the newer antibiotics have become generally available. A low residue diet is given. Heat in various forms is useful in diminishing spasm. Occasionally an ice bag is indicated by extreme soreness. Atropine and its derivatives, or other antispasmodics with small doses of a barbiturate sedative, are helpful. However, the barbiturates are often poorly tolerated by the aged. Jones²¹ advises barium sulfate by mouth twice a week to keep the diverticula filled. For many years it has been observed that the symptoms are improved after a barium enema.

Many of these cases have a diarrhea. In such cases, bismuth subcarbonate in doses of 20 to 60 grains three times daily will cause definite improvement. Bismuth subnitrate should be avoided because of the potentiality of nitrite intoxication as a side reaction. Care must be exercised to avoid constipation. If the stools become hard and difficult to expel, 3 to 4 ounces of warm cottonseed oil as a retention enema prevents impaction. Water enemas, if given at all, should be carefully injected under minimal pressure. Diverticula have been ruptured by enemas. Cathartics should be avoided.

CANCER OF THE COLON

Malignant disease of the colon increases in frequency with age. There is nothing else in the field of geriatric medicine which offers a better reward for a careful and thorough examination of the patient than colonic malignancy. Publicity given the large number of deaths every year from carcinoma of the bowel has helped to increase the public consciousness and fear of cancer. Every physician should be conscious of the possibility of a silent alimentary tract malignant tumor when examining individuals past forty-five, and should exhaust every procedure known to prove its presence or absence. The colon ranks a close second to the stomach as a site of gastrointestinal cancer.

Adenocarcinoma is the most common malignant tumor found in the large intestine. Metastases occur through the blood stream by direct extension, and by way of the lymphatics. The most common sites of metastases are the regional lymph nodes, liver, and lungs. It is desirable to seek routinely for evidence of metastases. Knowledge that metastases already exist alters the treatment indicated.

A continued and intensified effort is needed to effect earlier diagnosis. At present, the average time from the onset of symptoms to the diagnosis is approximately one year. Carcinoma of the right ascending colon causes symptoms later than does carcinoma of the left or descending colon. A sudden change in bowel habits in a patient past forty-five, without some easily determined cause, should lead the physician to make a complete diagnostic examination.

Symptoms. The right colon is large in diameter, and the contents are fluid in character. Thus obstructive symptoms appear late. The tumors in this region are frequently ulcerated, and absorption of toxic products is more com-

mon This leads to subjective symptoms of weakness, lack of the usual energy, and loss of appetite There is a slow loss of weight in the early stages The bowel movements may be formed, but constipation does occur, as well as diarrhea Gross blood is rarely seen in the stools, but chemical examination of the feces frequently reveals occult blood

A progressive anemia is very common, and should immediately arouse suspicion when occurring coincident with intestinal symptoms after forty Later a palpable mass appears in the right side It is not unusual to find that such a mass is the presenting complaint causing the patient to seek medical advice As a rule there is no true pain, but a feeling of fullness that is described as a distress or uncomfortable feeling This occurs in the right abdomen or it may be present in the epigastric region Food may or may not aggravate this distress It does not relieve it



Fig 116 Carcinoma of the ascending colon The only symptom was weakness with a marked anemia

In the left colon the fecal material is soft to firm in consistency, and the caliber of the colon is small The tumors that occur there are usually fibrous and have a tendency to grow around the intestinal wall, which makes the passage of the bowel contents more difficult A developing obstruction causes symptoms to be more pronounced Abdominal discomfort occurs much earlier than with right colon carcinoma At first the distress is not definitely localized, but as time passes, it becomes more severe and is localized to the left side Later this pain occurs at regular intervals gradually increasing in severity as the obstruction becomes more definite A complete evacuation of fecal material below the obstruction usually gives relief Still later a mass may be palpated, and peristaltic waves can be seen Usually by this time the pain is severe

The obstruction makes it difficult for formed material to pass and loose stools follow Constipation is not uncommon A diarrhea that continues for more than several days in the aged should arouse the suspicion of the clinician

that a serious lesion is present. Stools should always be examined for occult blood after the patient is given a meat free diet. A strong chemical reaction for blood will be found in most of the stools, and gross blood is not uncommon, as most of these lesions are in the rectosigmoid region.

Proctosigmoidoscopic examination gives valuable information when the carcinoma is in the rectosigmoid area, because it visualizes a part of the bowel that is difficult to visualize by roentgenoscopic examination. The rectal ampulla is usually markedly enlarged in old people. After a cleansing enema, and with the patient in the proper position, the examiner can frequently see the lesion. The well trained eye can make a diagnosis of malignancy. A biopsy should be made of any suspicious lesion. However, it should be remembered that the malignant portion may be missed and a negative report from the pathologist is not proof of absence of cancer. The clinician should not delay proper



Fig 117



Fig 118

Fig 117 Carcinoma of the cecum and ascending colon with all the findings and symptoms of carcinoma of the colon except obstruction.

Fig. 118 Carcinoma of the cecum and diverticulitis causing obstruction in the same individual

treatment if the biopsy specimen is found negative for cancer. If in doubt, exploratory surgery is more than justified. It is frequently possible for the examiner to see beyond the end of the proctoscope. Red blood may be seen flowing from a lesion higher up. In addition to the visualization of the lesion, fixation of the bowel can be determined. It must not be forgotten that the colon can be ruptured by the proctoscope.

The value of a *roentgenoscopic* and *roentgenographic* examination of the colon cannot be overemphasized. In most instances the use of castor oil before examination is undesirable, as it leaves the bowel highly irritated. However, if there is obstruction present and clinical evidence suggests an early carcinoma of the colon, a dose of castor oil the night before the examination will evacuate the fecal material. This will permit more effective visualization, aiding discovery of an early malignant lesion. Examination with a barium enema should reveal an obstruction, or what is more important, a persistent irregu-

larity in the contour of the colon. If roentgenoscopic study suggests a lesion in the region of the flexures, it may be necessary to take pictures in different positions in order to clearly reveal the lesion. The movability of the colon is also determined in this examination. If an obstruction is found, it is advisable not to get too much barium above the obstruction.

The physician should be alert to the probabilities of complications that occur in colonic malignancies. The most common are obstruction, fistula, ulceration, and perforation. Urinary complications may occur early and cause frequency of urination and dysuria. Pressure against the ureter may cause ureteral obstruction with its usual symptoms.

Diagnosis. Diagnosis of carcinoma of the colon depends upon the clinical history, a complete physical examination for palpable masses in the abdomen, and evidences of metastatic lesions. A chest x-ray should be routine, pulmonary metastases may be demonstrable before the original lesion is large enough



Fig. 119 Same patient as in Figure 118 after evacuation barium

to cause obstruction. It should be repeated that stool examination for blood is important. Anemia is typical, on the average the hemoglobin is lowest in the right-sided lesions, with a slightly higher hemoglobin if the lesion is left-sided. In the differential diagnosis, tuberculosis, diverticulitis, benign tumors, amebiasis, and chronic ulcerative colitis should be considered.

Treatment. The treatment is primarily surgical. The cooperation of an experienced surgeon, an internist, and a well trained anesthetist is necessary. The presence of metastasis and other complications may make extensive surgical procedure, such as resection, inadvisable. The type of operation to be done is best decided after the lesion and its complications are seen. Cancer surgery is radical surgery. If possible, a complete resection should be done.

When hepatic or other extensive metastases are revealed by surgical exploration, conservatism replaces radicalism. It is already too late to be radical. Colostomy above the obstruction, however, may prolong both relative com-

fort and life considerably. It must be remembered that, in general, the older the patient with neoplastic disease (either benign or malignant), the slower the growth of the lesion. Old people are better surgical risks than has been commonly believed. They should never be denied the potential benefits of surgery just because they are old.

Preoperative care is most important. Several days should be taken to prepare the patient for any colon operation. During this time, he should be put on a low residue diet in an effort to have the colon as empty as possible. Frequently these patients have put themselves on a limited diet of bland foods, and are deficient in vitamins, especially vitamin C. As mentioned before, it has been shown by Rafsky¹ that some vitamins are slowly absorbed by older people. Large doses of all vitamins should be given during this period. Protein deficiency, if present, should be built up by intravenous injection of amino acid preparations. A low prothrombin indicates the administration of vitamin K. The intake of sugar is helped by giving hard candies to hold in the mouth. Blood transfusions should be given to overcome anemia. If not contraindicated, small enemas should be given daily to keep the bowel below the lesion empty. Glucose and normal salt solutions are often indicated because of depletion due to vomiting. The amount of salt, protein, and glucose needed is best determined by blood chemical analysis and the degree and duration of vomiting. Such coincident complications as diabetes mellitus and prostatic enlargement, not uncommon in the aged, should be under control. The intake and output of fluids should be carefully observed. Sulfasuxidine is given for several days before the operation. It is desirable to administer a transfusion of whole blood during the operation. (See Chapter 8.)

The type of anesthesia should be determined by the anesthetist. Complete relaxation of the patient makes operation procedure easier and more rapid. (See Chapter 9.)

Postoperative care may require the use of Wangenstein suction, and in some cases the Miller-Abbott tube. It is well to remember that when gastric drainage is used a large amount of chlorides are lost, and that they must be replaced. It is advisable to remove the gastric tube as soon as possible. Proteins and vitamins should be continued, but intravenous administration must not be too rapid. The urinary output should be over 1000 cc. in twenty four hours.

If a colostomy is constructed, the patient should be taught how to care for it. In my experience, a colostomy has not been such a terrible thing as many patients anticipate. Most of the time it is not necessary to use a colostomy cup. Constipation can, if necessary, be encouraged by diet and medication until a single daily normal evacuation is routine.

Radium is not often used, as the lesions are not accessible. X ray therapy may have some temporary retarding influence when the malignancy has involved surrounding tissues or with recurrences. It is of particular value in sarcoma of the intestine.

Medical management of inoperable carcinoma consists of keeping the patient comfortable as long as possible, with all the moral support and analgesic medications available. Opiates should not be withheld too long. Keeping the patient happy and maintaining the morale of those most concerned is one of the most difficult tasks that come to the internist. The art of medicine takes over, for the science of medicine has been applied too late.

CHAPTER 35

DISEASES OF THE RECTUM

HARRY E. BACON

ANATOMY

Anal Canal. This is the terminal portion of the large intestine below the rectum. It begins at the anorectal line and ends at the anal orifice. It is lined with modified skin and is richly endowed with sensory nerve terminals.

Anal Papillae. These are small elevations varying in number from two to six, are slightly triangular in shape and of a pale pinkish color. The bases occur always at the anorectal line. Because of irritation these papillae may become inflamed and elongated.

Muscles. Surrounding the lower rectum and anal canal is the external sphincter muscle which is strung from the tip of the coccyx around the canal to be inserted into the perineum. The internal sphincter muscle is involuntary and represents an aggregation of circular fibers of the rectum.

Rectum. This is approximately six inches in length and extends from its junction with the sigmoid to the anorectal line. It is composed of mucous membrane, submucosa, and a muscle coat (inner circular and outer longitudinal). The rectum is invested on its anterior two-thirds by peritoneum forming the rectovesical or recto-uterine pouch. The blood supply of the rectum is derived from *superior* and *middle hemorrhoidal arteries* and of the anal canal from the *inferior hemorrhoidal arteries*. These vessels anastomose freely with the middle sacral artery. The *veins of the anorectum* begin as small venous sacs to form a huge plexus above the anorectal line. They are referred to as the *superior hemorrhoidal plexus* which drains into the portal circulation. Below the anorectal line the *inferior plexus* drains into the caval circulation.

The *lymphatics*, consisting of lymph capillaries, lymph vessels and glands, are well divided into an inferior group which drains into the inguinal nodes and the superior group which finally empties into the median lumbar glands.

The *nerve supply* of the rectum proper is sympathetic and parasympathetic, whereas the innervation of the anal canal and perianal region is somatic or cerebrospinal.

EXAMINATION AND DIAGNOSIS

History. All details as to pain, protrusion, bleeding, itching, swelling, discharge, and action of the bowels should be obtained in taking the history of a patient with an anorectal complaint.

Examination. This should be thorough, gently performed and should consist of inspection, palpation, digital examination, anoscopy, proctoscopy, and sigmoidoscopy. By *inspection*, skin tags, external thrombotic and varicose hemorrhoids, condylomata, the lower margin of a fissure, external opening or openings of a fistula or abscess, protruding internal hemorrhoids and prolapse may be noted. By *palpation*, a cordlike area of induration, representing a fis-

tulous tract, and the fluctuation and tenderness of an abscess may be disclosed. By the introduction of the *lubricated finger* gently into the anorectum, hypertrophied papillae, sphincter muscle atonia, not uncommon in later years, ataxia or spasm, deep crypts, longitudinal folds representing internal hemorrhoids, polypi, foreign bodies, and benign and malignant growths may be elicited. By *anoproctosigmoidoscopy*, fissure, hypertrophied papillae, internal openings of fistula, enlarged and infected crypts, internal hemorrhoids, mucous prolapse, foreign bodies, inflammatory and ulcerative processes, and malignant growths may be observed.

FISSURE IN ANO

Fissure in ano is an oval ulcer which appears as a slit and is usually placed vertically in the anal skin. It is of common occurrence and is encountered during the period of advancing years.

Trauma is the most important etiologic factor. It is brought on by the passage of hard stools, straining, insertion of foreign bodies, careless instrumentation and prostatic massage.

The pain itself is frequently excruciating and agonizing in character and the patient is generally wretched. The usual history is that following defecation or straining a stabbing pain occurs which lasts but a few minutes and then is followed by another seizure provoked by coughing, sneezing, crying, or motion. Bleeding is usually slight in amount, bright red in color, and occurs during or following bowel movement. Itching may or may not be present. Constipation is commonly associated and is due to fear of a bowel movement.

A history of a stabbing or lancinating pain of sudden onset associated with bowel movement, with the passage of a few drops of blood, is suggestive. The diagnosis is made on the presence of a slit-like ulcer, usually in the posterior midline of the anal canal. In the acute phase it is excruciatingly painful with associated muscle spasm.

Treatment. The pain in fissure in ano is due to exposure of the nerve endings and sphincter muscle spasm. For relief, the continuous application of compresses wrung out in hot witch hazel or boric acid solution is extremely helpful. Instillations of warm olive oil, 2 ounces, through a small rubber catheter or by means of a baby ear syringe are advantageous. Liquid petrolatum in small doses, two or three times daily, lubricates the stools and eases their passage through the anal orifice. It is recognized that excision of the fissure in its entirety is to be advocated as the procedure of choice.

CRYPTITIS

Cryptitis is an inflammation of the crypts of Morgagni which lie at the anorectal line between the bases of the columns. These are of importance because inflammatory processes therein are considered an important factor in the development of anorectal abscess and fistula.

Pain is common, oftentimes described as burning in character. A heaviness may be cited. Irritability of the sphincter is not uncommon, itching not relieved by scratching is frequent.

Excision of the crypts to the depth of the anal canal may be employed, but seldom with entire satisfaction. Excision or ablation of

the crypt may be done by the introduction of a few minims of procaine, 1 per cent solution, into the base of the crypt and when the crypt is drawn down the area can be excised

ABSCESSSES

Abscesses around the anorectum are relatively common and may be encountered in either sex and at all ages

Etiology. Infection through a crypt at the anorectal line, through the rectal wall from an ulcerative or strictured process or from some adjacent structure is the important factor. Use of sclerosing solutions in the treatment of internal hemorrhoids and mucous prolapse and the injection of various solutions beneath the perianal skin are occasionally followed by abscess formation

Classification Abscesses are classified according to their location. Those above the levator ani muscles include the pelvirectal, retrorectal and mural or interstitial, those below the levator (infralevator) are the ischiorectal and subcutaneous

Ischiorectal Abscess. This originates from an infected crypt

Symptoms are pain, tenderness, swelling, and heat on the affected side. The patient is unable to sit or lie down in comfort. Diagnosis is made on the presence of a swelling, redness, heat, tenderness, and fluctuation in the ischio-rectal fossa. The sphincter muscle is usually spastic

Treatment Immediate incision and drainage should be performed under some form of anesthesia or analgesia. In the office, ethyl chloride sprayed on the proposed line of incision or an intradermal injection of procaine may be used

TECHNIC Over the maximum point of fluctuation, the skin is painted with tincture of iodine and the skin infiltrated anteroposteriorly for a distance of 1 to 2 inches with a procaine solution, 1 per cent, employing a 25-gauge needle, to which is attached a 5 cc syringe. This proposed line of infiltration should be anteroposterior and about $1\frac{1}{2}$ to 2 inches in length, should be over the maximum point of fluctuation and should be just beyond the outermost fibers of the external sphincter muscle. The incision is then made by the scalpel to permit escape of the purulent material. The patient should be advised that this treatment is only an incision and drainage and that a fistula will result, which will necessitate an operative procedure at a later period (see p 557). A small wick of rubber dam, iodoform gauze, or gauze saturated with dichloramine-T may be loosely introduced for twenty-four hours. The wound should not be repacked thereafter. After-care consists of removal of packing at the end of twenty-four hours and the institution of hot sitz baths two or three times daily. If for any reason incision and drainage cannot be carried out, compresses wrung out in hot boric acid solution or witch hazel and/or hot sitz baths may be employed. In prescribing sitz baths the patient should be advised to sit in a tub of water six inches in depth, 110° F., (elbow heat) for three to five minutes, two or three times daily. In the aged it will be found that the application of compresses is less weakening to the patient than the tub baths. Ice packs should never be employed

Subcutaneous Abscess. This may arise from an infection of the perianal skin or as a residue from an ischio-rectal abscess

The patient usually describes a small swelling about the perianum that is tender to the touch and accentuated on assuming the sitting posture

Signs of an abscess confined to the immediate subcutaneous tissue will serve to make the diagnosis. If in the perineum, it will determine a perineal abscess and if immediately behind the anal aperture, a postanal abscess. In both of these a connecting tract invariably will be found at the anorectal line.

Treatment Incision and drainage in a longitudinal or criss-cross fashion providing the incision is made outside the outermost fibers of the external sphincter muscles is the procedure of choice. A small probe should be inserted posteriorly to determine if a tract extends from the anorectal line into a pocket. This may be materially assisted by the insertion of a curved probe or hook within the rectum. Dyes such as methylene blue are usually unsatisfactory. When the incision is made under procaine analgesia, similar to that described under treatment of ischiorectal fossa, the edges may be cut free. No packing is used.

Should the abscess recur, it is reasonable to assume that an undemonstrable tract from the anorectal line exists, in this case the patient should be hospitalized and the fistula dissected free to and including the internal opening. Low lumbar or sacrocaudal analgesia in the aged carries with it little chance of systemic reaction (see p. 158).

Pelvirectal Abscess. This may occur from an infected crypt, from the rectum proper, or from some adjacent tissue or structure.

Symptoms are obscure but a heaviness or weight in the pelvis or rectum is occasionally cited by the patient. Pain is experienced but does not approach the same intensity as that in an abscess of the ischiorectal variety.

With the finger in the rectum, a fluctuant mass involving the pelvirectal fossa laterally or anteroposteriorly is noted. The mucous membrane is movable over the mass.

Pelvirectal abscess may be distinguished from an intramural abscess by the fact that in the former, the mucous membrane is freely movable and in the latter, it is adherent, from retrorectal, in that this is always posteriorly placed.

Treatment As soon as the diagnosis is made, the patient should be admitted to the hospital because the surgical approach is entirely outside the rectum. An incision is made through the posterior portion of the ischiorectal fossa and through the levators. Like the retrorectal, but unlike the interstitial type of abscess, the approach should not be through the rectum.

Retrorectal Abscess. This usually arises from an infected crypt, extension of a postanal abscess, or from necrosis of the sacrum and coccyx.

The symptoms are somewhat obscure although the patient usually cites a heaviness and weight in the pelvis and low back pain with radiation down the sciatic nerve.

Digital examination reveals a fluctuant mass behind the rectum, over which the mucous membrane is freely movable. Deep pressure exerted over the skin between the tip of the coccyx and postanal verge is attended by considerable pain.

Treatment This requires hospitalization because it involves an extra-rectal approach through a diagonal anteroposterior incision with separation of the levators for drainage.

Intramural Abscess Ordinarily, this type of abscess begins from an ulcer-
 of the rectum. It may
 high

A feeling of fullness is usually cited and the abscess may give lumbar or urinary symptoms, depending upon whether it is located anteriorly or posteriorly

The fluctuant mass is confined to the wall of the rectum

Treatment In this particular abscess, the approach is through the rectum. Having located the abscess by digital examination, the scalpel is pressed against the gloved lubricated finger and introduced to the maximum point of fluctuation, a longitudinal incision $\frac{1}{2}$ to 1 inch in size is made. A better procedure is to introduce a speculum and, under direct vision, carry a longitudinal incision through the maximum point of fluctuation. A small drain may be introduced for a period of twenty four hours. Bleeding in these cases is usually negligible. The patient should consult the physician frequently thereafter, so that the cavity can be kept open and the base painted with gentian violet or some other aqueous solution.

FISTULA IN ANO

Fistula in ano (anorectal fistula) is a pathologic tract or abnormal communication between the anorectum and some adjacent tissue, viscus, or the skin surface.

Every fistula is preceded by abscess formation which is usually from an infection gaining entrance through a crypt of Morgagni, the rectum or adjacent tissue.

Varieties (1) Simple internal external, or internal complete or incomplete, (2) complex, where one or more internal openings with several tortuous and branching tracts and having multiple external openings exist. (3) complicated, when there is a tract from the anorectum, communicating with some diseased bone such as the sacrum or pelvis, or organ such as the bladder, urethra, uterus, vagina, or vulva. Encirclement of the rectum is termed "horseshoe fistula." It should be recalled that only approximately 2 per cent of all fistulae are tuberculous.

Symptoms include a history of preceding abscess, and persistent or intermittent discharge of pus, gas, or feces through an opening near the anus.

Diagnosis One or more small openings about the rectum from which pus may be expressed are present. These are often best revealed by a flexible silver probe, introduced with a finger in rectum as a guide. By palpation a cordlike tract is felt extending from the external opening toward a rectal crypt.

Treatment. This consists of complete excision of the tract together with the internal and external openings.

PILONIDAL SINUS AND CYST

(Sacrococcygeal, Sequestration, or Traction Dermoid)

This is a congenital defect resulting probably from faulty coalescence or invagination of the sacrococcygeal skin in the posterior midline during early embryonic development.

One or more openings occur in the midline or adjacent skin through which a tuft of hair may project. The sinus usually extends cephalad into a cyst. External manifestations are out of proportion to size of cyst cavity.

Symptoms are pain or discharge, with usually a history of trauma. Citation of previous abscesses opened spontaneously or incisionally is common.

Diagnosis is made on the presence of one or more openings in the dorsal midline of sacrococcygeal skin with sinus formation

Treatment Excision of sinus and cyst en bloc is the logical procedure The operation is ended in the open method by leaving the wound to granulate or primary suture may be done using alloy steel wire preferably

PRURITUS ANI

Pruritus ani is a syndrome embodying an alteration in the anal and perianal skin due to irritation of peripheral nerve endings caused by some local or systemic disease The chief symptom is itching of varying intensity which is characterized by chronicity and rebelliousness to treatment

Etiology Various causes may be mentioned such as alcoholism indiscretions in diet chronic disease of the liver and gallbladder poorly controlled diabetes mellitus vasomotor and endocrine imbalance but in almost every instance the causative factor will be found in the anorectum Of utmost importance are internal and external hemorrhoids hypertrophied skin tags papillitis and cryptitis Thread worms and various fungi also may give rise to itching

Symptoms Itching is the chief symptom and varies in intensity The patients are frequently wretched It is quite common following the act of defecation and is also prone to occur at night

Diagnosis The diagnosis is made on the history of itching and the appearance of the anal and perianal skin presenting thickening oftentimes denudation elongation and hypertrophy of the radiating folds

Treatment In each and every case all existing lesions should be removed Hemorrhoids should be treated by injection or operation fissures excised

calomel 2 grains given for three successive nights and rectal instillations of one of the following quinine bisulfate 1 2000 infusion of quassia 5 per cent or acetic acid 0.5 per cent solution Tinea trichophytina may be eradicated by the use of liquor carbonis detergens $\frac{1}{2}$ drachm to 1 oz or salicylic acid 15 grains to petrolatum 1 oz Locally a saturated solution of potassium permanganate may be painted on the surface daily while the patient may be given a prescription for the following

R ^y Phenol s	gr x
Lot on calam ne	q s 5

or

R ^y Sod i thiosulphat s	5i
Phenol s liquefact	5ss
Glycerini	5i
Aqua dest illata	q s ad 5vi
S g Apply locally	

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employment of an acid-ash diet, glutasin (glutamic acid hydrochloride) orally, and lactose suppositories.

Injection Methods. Various preparations for the relief of itching are used. . . .
instruments . . .

QUININE HYDROCHLORIDE. This is capable of producing prolonged analgesia. A site in the anterior commissure is sterilized, the skin of the anterior commissure painted with iodine, and a needle, to which is attached a syringe containing equal parts of procaine and quinine and urea hydrochloride each 0.5 per cent, is introduced. The solution is injected slowly beneath the skin until the entire pruritic zone has become slightly elevated. Ordinarily 15 cc. of the solution is employed. The treatment may be repeated at weekly intervals, if necessary.

ALCOHOL. Probably the best results are obtained with alcohol. In a series of cases there is a group in which marked abscess formation and prolonged convalescence occur, and used as prescribed it may be considered a failure. . . . If strict adherence to detail is observed . . .

The patient is placed in the Sims position and the skin prepared with tincture of iodine. A small wheal is raised by the injection of procaine on the left side just beyond the outer edge of the pruritic skin. A 22-gauge needle, three inches in length, is inserted through the wheal and advanced slowly under the skin. Ten cc. of 1 per cent procaine are injected subcutaneously, in a fan-shaped manner. The empty syringe is detached and, without withdrawing the needle, another syringe containing 10 cc. of 50 per cent alcohol is attached. The alcohol is slowly injected, moving the needle back and forth to distend the skin uniformly. Injection may be given on the opposite side five to seven days later and thereafter at weekly intervals, if necessary. If the skin becomes markedly inflamed, hot boric acid compresses should be applied continuously or the patient may be instructed to sit in six inches of water at elbow heat, for five minutes.

HEMORRHOIDS

Hemorrhoids are varicose dilatations involving one or more radicles of the hemorrhoidal veins.

Etiology. Any cause which brings about a retarded return circulation is conducive to hemorrhoidal disease. In the period of advancing years muscular atonia offers diminished support which makes the already existent hemorrhoids easy of protrusion. Constipation and the distention and pressure exerted by accumulated hard fecal masses are frequent causes. Engorgement of the portal circulation, increased abdominal pressure from tumors, corsets, or belts, mucosal irritants, and drastic cathartics are important causes. Infection, of course, is the most important factor to be considered.

Symptoms and Diagnosis. An external thrombotic hemorrhoid is an oval swelling, usually single, noted in the region of the anus and is usually tender, firm, and of bluish color. The symptoms are of sudden onset and the pain of a throbbing character, aggravated by sitting, walking, or defecation. The diagnosis is made by the sudden onset and the presence of a single, firm bluish swelling.

Treatment. Mineral oil by mouth, a bland diet, compresses wrung out in hot boric acid solution, and sitz baths for five minutes at 110° F. in six inches

of water are to be advocated Ointments are not very satisfactory, although the following may be employed

R̄ Ichthyol	gr xxv
Anesthesine	ʒss
Argyrol	ʒi
Petroleum jelly	q s ad ʒi
Sig Apply locally	

or

R̄ Ung stramonii	ʒiss
Ung belladonnae	ʒiiss
Ung acidi tannaci	ʒss
Sig Use in and outside anus	

Excision A few minims of a 1 per cent procaine solution are injected into the skin over the hemorrhoid by means of a fine, sharply pointed needle By injecting the solution in the line of the proposed incision and into the layers between the skin and the clot, the pile mass will be made to appear swollen A single incision is made in the longitudinal axis from within out and curved scissors introduced to separate the fine fibrillations between the undersurface of the skin and the clot itself The clot is then expressed Bleeding is negligible The after treatment consists of hot sitz baths and the topical application of gentian violet, 1 per cent

Cutaneous Hemorrhoids or Skin Tags These are found at the margin of the anus and occur as flabby redundancies In elderly individuals they are quite common

Previous thrombosis is the most important cause

Transient itching is not unusual When the tags are inflamed tenderness may be mentioned

Treatment Cleanliness freedom from toilet paper irritation and a soothing ointment, such as the following, may be used to advantage

R̄ Ichthyol	ʒiv
Ung belladonnae	
Ung stramonii	ʒa ʒi
Sig Apply locally	

Gentian violet, in 1 per cent aqueous solution should be applied to the surface once or twice daily Occasionally, a powder of calomel 1 drachm and bismuth subcarbonate 3 drachms is helpful

Excision is indicated where inflamed tags have not responded to palliative measures One half to 1 cc of 1 per cent procaine solution is injected into the base of the tag which is grasped with hemostats An incision is carried around the base and one to two sutures are introduced Mercurochrome, 5 per cent, or gentian violet, 1 per cent is painted on the surface daily The sutures are removed on the third postoperative day

Internal Hemorrhoids Bleeding and protrusion are the most common

Diagnosis is made on proctoscopic examination which discloses one or more globular masses covered by mucous membrane

Treatment This comprises regulation of the bowels by the use of liquid petrolatum, 3 to 4 drachms, once or twice daily. Instillations of ichthyol, warm water or olive oil, or witch hazel are of value. An ointment of the following may be employed to advantage

℞ Ext belladonnae	gr ⁱⁱⁱ
Petrolatum	5i
M et ft unguentum	

Sig Apply locally

Strangulated Hemorrhoids These are best treated by continuous application of hot boric acid compresses, which diminishes the edema. Strangulated hemorrhoids should never be operated upon in their acute phase. Immediate reduction under gentle taxis and continuous application of the compresses should be instituted.

INJECTION TREATMENT OF HEMORRHOIDS

The treatment consists in the injection of a sclerosing solution into the submucosa overlying the hemorrhoidal veins to incite the formation of fibrous tissue, which obliterates the varicosities of the internal pile mass. The procedure is especially suited to patients of advanced years.

Indications and Contraindications Small or moderate sized and uncomplicated internal hemorrhoids may be treated by this method. It is inadvisable to use the injection treatment in the following: (1) external hemorrhoids and skin tags, (2) marked fibrosis, (3) adjacent disease, such as fissure, fistula, cryptitis, tumors, proctitis, or phlebitis, (4) inflammation and prolapse whether associated or not with thrombosis, strangulation, or ulceration, (5) a spasmodic sphincter muscle which indicates the presence of infection.

Advantages In skilled hands, the treatment causes no pain. The method requires neither anesthetic nor hospitalization. The patient suffers no disability and finds it economical. The procedure can be utilized without risk in diabetics, in old age, and in patients with cardiac, pulmonary, or renal diseases.

Disadvantages The treatment is limited to a very few selected cases. The frequency of recurrence is relatively high. Indiscriminate injection of hemorrhoids, with insufficient knowledge of the technic and of the anatomy of the anorectal region, is fraught with dire consequences for the physician and the patient.

Armamentarium Good lighting, whether direct or indirect, is of utmost importance. Any type of syringe may be utilized. A 5 cc durable glass, inexpensive syringe should be employed for an oil solution, and a small tuberculin syringe will be found of service for the quinine and urea hydrochloride solution. The physician must use the syringe which he handles most conveniently. A needle that is sharp, of medium bevel and 24- or 25 gauge is preferable, although a larger gauged needle will be found more suitable for an oil solution. A specially crooked neck extension or even a double curvature needle will permit perfect vision and proper injection. The physician must be guided in his choice of a speculum by no fast rule. The one to which he has become accustomed is usually the most appropriate. Ordinarily a Martin proctoscope is

employed Only three solutions will be mentioned, in the order of their serviceableness, a 5 per cent solution of quinine and urea hydrochloride, a 5 per cent solution of phenol in oil, preferably a vegetable oil, such as olive oil, and a 5 per cent solution of sodium morrhuate in benzyl alcohol

Technic. The patient lies on his left side, the right buttock is raised and examination of the anal canal is made with a well lubricated finger In turn, the speculum is smeared with a water-soluble lubricant and is gently inserted with a circular motion

The hemorrhoid to be injected is located, its surface is cleansed with a cotton applicator and daubed with a 5 per cent solution of mercurochrome The needle is then introduced in the submucosa and the required amount of solution is injected

Certain rules govern the injection (1) The first injection is made at the upper pole of the hemorrhoid A low injection is not only apt to cause pain, but fibrosis will occur in this region and will render the treatment ineffective so far as the disappearance of the hemorrhoidal mass is concerned It is better to work downward as the treatment progresses

(2) The injection should not be given in the mucosa proper nor in the veins A snow-white spot will reveal the former, bleeding will give evidence of the latter The injection in this event should cease immediately and another site should be chosen Sloughing with its sequelae of ulceration, proctitis, abscess, fistula, and the like, is apt to result from poor technic

(3) Pain is caused by an injection given too near the anorectal line

(4) No force must be exerted on the plunger When a sufficient amount of solution has been injected, resistance is offered and this should be recognized by the operator

(5) An inexperienced operator should limit himself to very small amounts of solution Otherwise the amount may vary from a few minims to 2 cc Best results are obtained when ballooning is observed The mucosa then has the appearance of a very light parchment presenting venules and arborizations

(6) Care must be taken to use fresh solutions Much of the discredit of this method is due to the negligence of the operator in not mixing the solutions well or in not renovating old stagnant solutions

The number of injections varies with the individual and a full course of treatment comprises from six to eighteen injections

The injections are given at intervals of four to seven days and in a clockwise pattern Success is obtained only in cases that are treated in regular, sustained, closely followed fashion One or two injections may be given at one time, but when two injections are given, opposite sites are chosen If necessary, a second injection may be given in a previous site, but only after the induration subsequent to the first has *entirely* disappeared

Care Following Injections Discomfort and the feeling of pressure when present are relieved by the use of hot sitz baths and hot compresses Pain is the result of faulty technic or of abscess formation A longitudinal incision in the axis of the bowel over the site of the injection will procure instant relief The patient is instructed in the proper evacuation of his bowel Liquid petrolatum, agar-agar, or milk of magnesia should be prescribed as the occasion demands The patient is also instructed to replace any protrusion of the pile mass that should occur after the injection

The best treatment for all varieties of hemorrhoids is surgical removal be

cause if properly performed a permanent and good result is achieved. Standard methods such as excision and suture are to be recommended.

PROLAPSE AND PROCIDENTIA

Both of these conditions are encountered with relative frequency in elderly individuals as they are due to muscle and fascial relaxation. Prolapse is characterized by the abnormal descent of the mucous membrane of the rectum, whereas procidentia connotes the abnormal descent of the entire bowel wall, namely the mucosa, submucosa, and muscle coats. The treatment of the latter is distinctly surgical, for which the reader is referred to any treatise on proctology.

Etiology Straining at stool, such mechanical factors as polypi, hemorrhoids, and hard costive stools, and inflammatory processes, such as coloproctitis, are important factors.

Diagnosis Diagnosis is made by the history of protrusion at stool or some effort at straining. Inspection discloses a spherical mass of bright red color with radiations from the center. Procidentia is differentiated by the fact that the striations are circularly arranged. Protrusion of the rectum, sensations of pressure or weight in the pelvis, fullness or bearing-down sensation, are frequent complaints.

Treatment All factors conducive to straining should be eliminated. Liquid petrolatum by mouth may be given nightly. The diet should be highly nutritious, leaving but little residue.

Injection Therapy with Quinine and Urea Hydrochloride The injection of quinine and urea hydrochloride has proved beneficial and is worthy of trial in mucosal prolapse. With the patient in the left lateral position, a conical speculum is introduced and 15 to 20 minims of 5 per cent solution of quinine and urea hydrochloride are injected into the submucosa in each quadrant of the prolapse. The treatments may be repeated at weekly intervals and reexaminations should be made periodically thereafter. Do not inject the prolapse outside the anal aperture.

The extrarectal injection of hydrochloric acid has proved of value in prolapse but not in procidentia. An antiseptic is applied to the skin over the right ischiorectal fossa and the pararectal structures infiltrated with 30 to 40 cc. of 0.5 per cent procaine solution. The needle, $3\frac{1}{2}$ inches in length, is advanced

surgical correction should be undertaken. Low lumbar and especially sacrocaudal analgesia may be employed at any age with little or no danger.

Procidentia may be corrected only by an abdominal approach utilizing fixation, plication, shortening lateral ligaments and, in the female, uterine suspension.

MALIGNANCY OF THE ANUS, RECTUM, AND SIGMOID COLON

Certainly a larger number of cases of cancer involving these parts are being recognized, but it is the consensus that this is due to improvement in diagnostic technic and more widespread use of these methods, together with

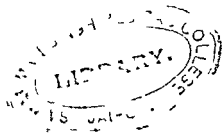
the fact that patients are carried over the gaps of intermediary illnesses better and therefore reach an age when cancer is more frequent. The vast majority are encountered between the ages of forty and seventy. Males are affected more frequently than females. According to our records, cancer in this locality represents approximately 80 per cent of all malignant tumors in the intestinal tract and 12 per cent of all such malignant growths in the human organism. The most common variety in the rectum and sigmoid is adenocarcinoma, and of the anal canal, epithelioma or skin cancer.

Symptoms Every elderly patient presenting a history of bleeding from the bowel or change in bowel habit, must be considered as having a potentially malignant lesion until proved otherwise by all the available means of diagnosis. Quite true it is that there are no pathognomonic symptoms of cancer. *Highly suggestive*, however, are change in bowel habit—such as progressive constipation or constipation followed by numerous stools, early morning diarrhea, frequent desire for stool, at which time the patient expels little other than flatus, mucus, and blood. *Pain and loss of weight* are late symptoms. Pain from cancer of the rectum never occurs except when it exerts pressure on the sacrum, bladder, or prostate or by extension and metastasis. With epitheliomata, on the other hand, pain is usually extreme.

Diagnosis In making a diagnosis of cancer, every means must be resorted to, such as thorough digital and proctosigmoidoscopic examination, biopsy of tissue and, for lesions of the sigmoid, roentgenographic study following an opaque enema. The diagnosis is made on the presence of a new growth, more frequently above the hemorrhoidal area but usually within reach of the finger, which is fixed, hard, and nodular. Through an illuminated proctosigmoidoscope the mass appears like a cauliflower, bleeds easily, and is friable. Usually a deep excavating ulcer is present and shows a deep necrotic base with everted, rolled, and nodular edges. A biopsy specimen confirms the diagnosis.

Treatment The only treatment for cancer of the anus, rectum, and sigmoid colon is radical surgical extirpation. It is indeed surprising to observe how well aged patients progress following major surgical removal, if they have been hospitalized for several days preoperatively to receive intravenous fluids and blood with especial attention to cardiovascular, pulmonary, and renal systems. Fractional spinal analgesia has proved ideal in the aged. In a personal series of over 800 patients with malignancy of the lower bowel, over 50 per cent are alive and well following resection. As to technic, it has been our experience that an abdominal colostomy is nearly always indicated in the presence of an acute colic obstruction, but seldom in the radical removal of the cancerous process. Proctosigmoidectomy without colostomy and with preservation of the sphincter muscles has proved its merit.

Deep x ray therapy should be reserved for inoperable cases where metastases are present and for the control of intractable pain.



CHAPTER 36

DISEASES OF THE LIVER AND BILIARY PASSAGES

GEORGE MORRIS PIERSON

LIVER

It is generally recognized by students of geriatrics that few, if any, diseases are primarily the result of old age. In the old, as in younger individuals, disease is the result of disturbed environment, changed by such factors as infection, fatigue, trauma, malnutrition, and others. Old age plays a contributory role, modifying, delaying, or accelerating disease processes. The problem which confronts the gerontologist is to determine the susceptibility of the aging organism to various diseases and how, when they occur, their course, clinical manifestation, prognosis, and management are modified and influenced by age. To arrive at a proper understanding of this, it is necessary to learn how the aging process alters both structure and function and what effect such changes have upon reaction to disease, not only of the body as a whole, but of any organ under consideration.

The liver is no exception to the general principle just set forth. Broadly stated, there are no diseases of the liver peculiar to or solely dependent upon old age. The liver of an old individual may be subject to the same pathologic processes that are encountered in younger persons, although the frequency and clinical course of these are definitely modified by the age factor. Properly to evaluate the variations which occur, it is of advantage to inquire into physiologic and anatomic changes that are brought about in the liver by age itself.

PHYSIOLOGICAL CONSIDERATIONS

Decrease in Weight. It is generally agreed that, other things being equal, there is a weight loss in all organs in old age. The liver conforms to this rule. The human liver increases in weight from birth until the age of twenty. During

the age of seventy five years has decreased to an average of 1480 gm. She found that in white females the median weight of the liver had decreased to 1180 gm. by the age of seventy-five. This normal decrease in liver size, which may be attributed to aging, in actual experience is frequently offset by enlargement of the liver, secondary to circulatory changes and degenerative states such as fatty degeneration.

It is often stated that with advancing years the connective tissue stroma of the liver increases. This observation has been disputed by Frischmann,² who carried out histologic studies on the effect of aging on the connective tissue of the human liver. It is his contention that the increase in connective tissue so frequently found in the livers of older people is not due to physiologic aging but is the result of various pathologic processes. This was borne out by

his failure to find any increase in the connective tissue of Glisson's capsule or in the intercellular connective tissue of the liver of ten subjects aged from sixty to eighty-six years

Functional Activity. In spite of the physiologic decrease in weight that occurs as age advances, the functional activity of the human liver, as measured by ordinary liver function tests, does not decrease with age, according to Ivy³ He considers it not surprising that this decrease in liver weight should not be accompanied by liver insufficiency, when one takes into account the large factor of safety possessed by the liver In support of this, he cites the work of Mann and Magath⁴ who showed that four-fifths of the liver of an adult dog can be removed before impaired liver function can be demonstrated by the ordinary liver function tests He also recalls the extraordinary ability of the liver to regenerate, as was shown long ago by Whipple and Sperry⁵ following the production of liver cell necrosis with chloroform

There are certain hepatic functions that appear to be inhibited or decreased as the result of age *per se* The *glycogen content of the liver* at different ages was studied in the rat by Deuel, Butts, Hallam, *et al*⁶ They found that the liver glycogen increased after birth up to 8 per cent when the animals were forty days of age, after which it gradually declined to about half of that value in rats that were one and one-half to two years of age According to the observations of Yadorsky, Almaden, and King,⁷ the vitamin C content of the liver decreases as the individual's age advances

It is highly probable that senile liver cells possess a *greater resistance to hepatotoxic substances* than do the hepatic cells of young individuals This is borne out by the observations of MacNider⁸ in his extensive studies on the acquired resistance of fixed tissue cells When he subjected old dogs to experimental chloroform poisoning, he found that their livers manifested a greater tolerance to the hepatotoxic agent than did the livers of younger animals Furthermore, the older livers reacted normally to the phenoltetrachlorophthalin clearance test

Although the liver is the largest gland in the body and exercises many complicated and important functions essential to the animal economy, surprisingly little data are available upon the effect of aging on the physiology of this organ The paucity of our information in this field is another gap in our knowledge of geriatrics and presents many opportunities for profitable research

CIRCULATORY DISORDERS

Chronic Congestion. As has already been suggested, there is no justification to ascribe particular diseases to old age, on the other hand, many well-recognized hepatic disorders are frequently encountered in the aged Chronic, passive congestion of the liver is the circulatory disturbance of the liver most often observed in older persons It is commonly the *result* of chronic right-sided cardiac failure which, in older people, is due to myocardial degeneration from defective nutrition of the heart muscle In older people this is the result of coronary sclerosis, repeated coronary occlusions, or states of general malnutrition (see p 412) More rarely, the right-sided cardiac dilatation is the outcome of a chronic, adhesive pericarditis or some form of pulmonary obstruction such as emphysema, chronic bronchitis, bronchiectasis, widespread pulmonary fibrosis, or chronic adhesive pleurisy (see p 316) In exceptional cases, chronic congestion of the liver is the result of obstruction of the inferior

vena cava above the entrance of the hepatic vein. Such obstruction may be due to a tumor, aneurysm, or glandular enlargement.

In the early stages of passive congestion, the liver is enlarged, firmer than normal, and purplish red in color. On section, it presents the well known nutmeg appearance due to the congestion which begins about the central vein of the liver lobules and gradually spreads towards the periphery. As the congestion continues, a gradual overgrowth of connective tissue takes place, giving rise to a condition that has been termed cardiac cirrhosis.

In addition to the *symptoms* of the underlying disorder, those that are referable to the liver consist of pain, tenderness, and a sense of fullness in the right hypochondrium and epigastrium. At times slight and variable degrees of jaundice may be noticed. On *physical examination* the liver is found to be enlarged, it is frequently tender, uniformly smooth, and the edges are sharp. Occasionally when a tricuspid insufficiency exists, an expansile pulsation of the liver may be elicited. In 235 cases of tricuspid regurgitation studied by Pitt⁹ a true pulsation of the liver was observed in fifteen.

There is no special treatment for chronic passive congestion of the liver other than that of its underlying cause.

Pylephlebitis Inflammation or obstruction of the portal vein or its branches may occur. There is no reason, however, to believe that this condition is any more frequent in old age than it is in earlier life, or that its clinical manifestations are modified by age. Acute pylephlebitis arises from acute inflammation in the areas drained by the portal vein. Bacteria or infected thrombi are carried toward the liver and set up inflammation in the portal vein or some of its main radicles. The commonest *causes* of pylephlebitis are acute appendicitis, inflammations in the hemorrhoidal area and genital organs, ulcerations of the intestines, particularly dysentery, ulcerating neoplasms

the severe general infection of which they are a part. The condition is usually associated with the signs of sepsis, such as hectic fever, chills, sweats, prostration, and leukocytosis. Symptoms that suggest involvement of the portal vein are rapid enlargement of the spleen and the occurrence of ascites. The *prognosis* is always grave. Before the advent of the sulfonamides and penicillin the results of *treatment* were almost uniformly unsuccessful. If the underlying focus of infection can be removed, the adequate use of sulfa drugs and penicillin or some other appropriate antibiotic, depending upon the type of causative organism, now offers some hope of recovery.

Portal Vein Thrombosis *Thrombosis of the portal vein or its branches* may occur from compression by glands in the region of the liver, carcinoma of the pancreas, malignant disease of the bile duct, distortion of the veins due to cicatricial contractions, the result of chronic inflammation, or the extension of a malignant growth into the vein.

The *symptoms* of such a thrombosis are those of portal obstruction which develops with more or less rapidity. They vary according to whether the main vein has been obstructed or only some of its radicles. The severity of symptoms and their time of onset depend upon the completeness and the rapidity with which the portal vein is obstructed. If the thrombosis is incomplete and slow in formation, the development of collateral circulation delays the onset

of marked symptoms. When longstanding complete obstruction of the main branch of the portal vein occurs, it is marked by splenic enlargement which often attains considerable size, the development of ascites which rapidly recurs after paracentesis, disturbances of gastric digestion, hematemesis due to dilated gastric and esophageal veins, intestinal hemorrhages, dilated hemorrhoidal veins, and prominence of the veins over the abdominal wall and the lower portions of the chest, including those about the umbilicus, which at times gives rise to the so-called caput Medusae. As the result of gastrointestinal bleeding, a considerable degree of anemia may be present. Sometimes thrombosis of the mesenteric vessels brings about hemorrhagic infarcts of the bowel and subsequent peritonitis.

The *prognosis* depends upon the underlying cause. In the majority of cases the outlook is unfavorable unless the obstruction can be relieved. When infection coexists the employment of the sulfonamides and penicillin in conjunction with supportive measures offers some added hope.

DISEASES OF THE LIVER

Acute Hepatitis. Acute hepatitis is a general term used to designate a number of acute and subacute conditions of the liver, due to a variety of etiologic factors, which are characterized by jaundice of hepatocellular origin and its associated clinical features. Acute hepatitis may be divided into two main groups: infectious hepatitis and toxic hepatitis.

Under the first head are included: (1) Acute bacterial hepatitis in which the liver is the seat of primary bacterial invasion or is secondarily involved as a complication in various septicemias, typhoid, pneumonia, and other infectious diseases; (2) Infectious leptospiral hepatitis (Weil's disease), a specific infection due to the *Leptospira icterohaemorrhagiae*; (3) Amebic hepatitis, a complication of amebic dysentery; (4) Infectious viral hepatitis, variously referred to as icterus simplex, epidemic hepatitis, epidemic jaundice, infectious jaundice, or by the older and most commonly used term catarrhal jaundice.

Although the specific etiologic factor in the last named group is still uncertain, the weight of evidence points strongly to a filtrable virus as the exciting cause. In this category should be included epidemic infectious jaundice, so frequently encountered in our armed forces during the recent war, as well as those cases of jaundice which follow injections of sera or vaccines or the transfusion of pooled plasma and blood.

Since all forms of infectious hepatitis are found chiefly in children and young adults they merit little consideration in a discussion of the liver disorders peculiar to old people. In a review of 200 cases of infectious hepatitis studied at the Hospital of the Rockefeller Institute by Hoagland and Shank,¹⁰ only 1 per cent were in patients aged over forty years. On the other hand, all forms of infectious hepatitis can and at times do occur in older individuals. This is especially true of the infectious viral variety when it appears in epidemic form, thus giving rise to serious diagnostic difficulties.

Toxic hepatitis results from ingestion of or other exposure to a number of drugs and chemicals. The drugs most often responsible for liver damage are arsphenamines, the sulfonamides, and cinchophen and its derivatives. Of the chemicals that cause injury to the liver, many of which are of great industrial importance, may be mentioned alcohol, ether, chloroform, trinitrotoluene, dinitrophenol, carbon tetrachloride and other chlorinated halogens, picric

acid, phosphorus, arsenic, lead, gold, bismuth, and copper. To this list may be added snake venom, poisonous fungi, and incompatible blood and hemolytic sera.

In the older patient acute hepatocellular degeneration is more often of toxic than infectious origin. Of the above enumerated toxic agents those most commonly responsible for liver degeneration in old people are the arsenicals, the cinchophen derivatives, and the sulfonamides.

Arsenical hepatitis usually occurs during the treatment of syphilis with some of the arsenical preparations. Most investigators agree that the arsphenamines are much more toxic than the inorganic preparations of arsenic. Individual sensitivity plays a large role in the development of this type of hepatic disease. States of malnutrition and previous liver disorders, such as cirrhosis, are probably important contributory factors. According to Sager¹¹ the occurrence of jaundice bears a direct relation to the amount of arsenical used. On the other hand, Bockus and Tumen¹² have seen a toxic hepatitis develop after two injections of neoarsphenamine totalling only 0.6 gm. The symptoms may come on from one day to two weeks after the drug is first given. Delayed cases, according to Wile and Sams,¹³ develop about three months after treatment has been started.

Acute hepatocellular disease from *cinchopen* or some of its derivatives, although less frequently encountered generally than arsenical hepatitis, is met with more often in old people than is the latter. Active arsenical treatment of syphilis is less likely to be carried out in older persons, but they frequently suffer from various muscular and arthritic disabilities loosely classified as gout or arthritis, for which cinchopen is either administered or taken on the initiative of the patient. Liver injury due to cinchopen is apparently not related to the amount of drug taken. Here again individual susceptibility seems to be a determining factor. Jaundice may occur soon after the drug is administered or may not develop until three weeks after its discontinuance.

Sulfonamide Jaundice The chief symptom which indicates liver disturbance during or following the administration of a sulfonamide is icterus. Jaundice may be produced in two ways, either by direct injury to the liver cells which results in a toxic hepatitis or as the result of an acute hemolytic crisis and severe anemia. In some instances both mechanisms are operative. According to Long and his coworkers^{14, 15} jaundice as a toxic manifestation of sulfonamide therapy is more often due to anemia and a hemolytic crisis than to hepatitis. The same observers found sulfanilamide the preparation most often responsible for hepatitis and hemolytic crises. Even when clinical jaundice is absent evidences of impaired liver function have been observed after sulfonamide therapy.¹⁶

As is the case with other drugs, there is considerable variation in the amount of sulfonamide that will cause liver damage. Jaundice has been observed following a single dose, whereas in other instances it required 50 gm. before evidence of hepatitis appeared.

The prognosis of sulfonamide hepatitis as a rule is good, the condition rapidly subsiding after withdrawal of the drug. On the other hand, in a few instances acute yellow atrophy of the liver has developed rapidly. Whether toxic cirrhosis in later life will develop in livers that have been damaged by sulfonamides is not yet known.

Symptoms The symptoms of acute hepatocellular disease, whether it is

due to an infectious or a toxic agent, follow much the same course. Anorexia, nausea, and vomiting, usually associated with abdominal pain, weakness, and languor, are followed in a few days by jaundice which may be slight and fluctuating or severe and progressive. The liver is usually enlarged, sometimes there is tenderness and in a certain proportion of the cases the spleen is palpable. In the milder form, the jaundice gradually subsides and the patients recover in the course of some weeks. In the severe cases, however, the jaundice becomes progressively worse and headache and delirium occur, often accompanied by high fever and chills. The urine is suppressed, severe toxemia develops, coma and death terminating the process. In short, the final picture is that of hepatic insufficiency as seen in acute yellow atrophy. Some of the cases that survive the acute stage develop a subacute necrosis, which runs a prolonged course terminating in that form of chronic hepatitis which has been referred to by Mallory as "toxic cirrhosis."

Diagnosis The diagnosis of acute hepatocellular disease is often difficult. It is important from the standpoint of treatment to differentiate the jaundice due to destructive liver disease from that which results from obstruction of the bile ducts. In this, certain liver function tests are of decided value. A positive galactose tolerance test, if done early in the course of the disease, is fairly conclusive evidence of parenchymal liver damage as the cause of jaundice rather than biliary obstruction. The blood phosphatase is normal when icterus is of the nonobstructive type. If jaundice is the result of liver cell disease, the total blood cholesterol is normal or decreased and the percentage of cholesterol ester is definitely decreased. In severe cases, the ester may entirely disappear from the blood. Hippuric acid excretion is normal in obstructive jaundice and decreased in parenchymal liver disease.

Treatment In the treatment of acute hepatocellular disease, it is essential to eliminate the offending drug or other source of intoxication or infection at the first appearance of jaundice. The most important factor in successful treatment is the institution of absolute *bed rest* as promptly as possible after the diagnosis is made. Restricted activity should be insisted upon until convalescence is well established. Not until all objective evidence of disease has disappeared and laboratory tests show no residual liver dysfunction should patients be allowed to go out. The beneficial effect of a rigid regimen of rest on the duration of convalescence from infectious hepatitis was clearly demonstrated by Hoagland and Shank.¹⁷

Proper *diet* is the next most important feature of treatment. Since the best safeguard against liver cell damage is an adequate glycogen store within the liver, a diet high in carbohydrates is essential. If possible 300 to 400 gm. of carbohydrate should be given daily. This can be accomplished by adding lactose and glucose to the diet. If the requisite amount of carbohydrate cannot be taken by mouth or the patient is acutely ill, it should be introduced by slow intravenous injection of 10 per cent glucose solution. An adequate fluid intake by mouth or parenterally should be assured. It has been shown that the regeneration of liver cells is stimulated by the ingestion of adequate protein. Therefore, the daily diet should contain from 80 to 120 gm. of easily assimilated protein. Fats, because they are poorly digested in the presence of jaundice and because of their deleterious effect on a damaged liver, should be sharply reduced during the acute stage of the disease and even after convalescence is well established.

The so-called hydrocholeretic drugs, which frequently do more harm than good, should be avoided. In severely toxic patients especially if hemorrhages occur, blood transfusions are of value.

Liver Abscess. Liver abscess or suppurative hepatitis is less commonly encountered in senescence than in earlier life. There is nothing distinctive about liver abscess in old people. It may be single or multiple. The *single abscess*, often called tropical abscess, is almost invariably a complication of dysentery and is due to the *Endamoeba histolytica* so commonly found in the tropics but also met with in the temperate zone as well. Tropical abscess is rarely found in children or in the old, most cases occurring in those between the twentieth and fiftieth years. Between 60 and 80 per cent of such abscesses involve the right lobe and are usually located in the superior posterior portion.

Multiple abscesses of the liver may be the result of trauma, a bacteremia or the extension to the liver of a contiguous suppurative process such as an

adult life, since this is the age period in which those infections most likely to give rise to such abscesses have the greatest incidence.

The *symptoms* of liver abscess may vary considerably. Single tropical abscess may run a protracted course and be unrecognized for a long period of time. The symptoms of multiple pyogenic abscesses vary with the underlying cause and the virulence of the infecting agent. Frequently the initial disease masks the symptoms of liver abscess. The more acute forms of liver abscess are characterized by the presence of irregular fever, frequently chills, leukocytosis, pain over the liver, enlargement of the liver, occasionally jaundice, and tenderness over the liver which can be brought out as a rule by Murphy's fist percussion. X-ray examination will usually show elevation of the diaphragm and restriction of movement on the affected side.

and tertiary stages of the disease, the average age incidence of which is between thirty and fifty years.

The only form of syphilis of the liver that is of interest to the student of

subdivides the lobes of the liver into numerous irregularly shaped lobules of varying size, giving rise to a distorted and grossly irregular liver. When *symptoms* of true syphilitic cirrhosis exist, they are dependent entirely upon the mechanical effect of the bands of connective tissue which subdivide the liver. As the result of pressure these bands may bring about obstruction of the portal vein, with its symptoms of portal obstruction including ascites, on the other hand, biliary obstruction may occur. In most instances the condition progresses slowly with no definite symptomatology. On *physical examination*, a markedly irregular and nodular liver may be felt.

Portal Cirrhosis The chronic types of liver disease are more important from the standpoint of senescence than the more acute conditions that have just been discussed. The form of chronic liver disease that is most often met with in old people is that variety of chronic hepatitis known as portal cirrhosis. In the literature, this variety of cirrhosis of the liver is frequently referred to as Laennec's, atrophic, or alcoholic cirrhosis. The term "portal," however, is preferred because it is more descriptive of the condition, since the toxic agent, whatever it may be, is conveyed through the portal vein and its branches, and the most conspicuous symptoms are those associated with obstruction of the portal blood flow.

The frequency of cirrhosis of the liver in old people may be gathered from the vital statistics published in 1940.¹⁸ According to this report, in the United States during 1937 there were 10,808 deaths ascribed to cirrhosis of the liver of all varieties of which about two thirds occurred in males, the greatest number of deaths occurred between the ages of fifty-five and seventy-nine. In Mallory's series, reported from the Pathological Laboratory of the Boston City Hospital,¹⁹ there were 487 cases of cirrhosis of the liver, of which 248 were of the portal variety. Of this number, sixty-eight occurred in the sixth decade, thirty-six in the seventh, ten in the eighth, and two in the ninth decades, making a total of 116. Thus portal cirrhosis was demonstrated after the age of sixty in about half the group. As a rule, portal cirrhosis develops during the fourth and fifth decades. Although, for the most part, portal cirrhosis begins in middle life, the disease progresses slowly, especially in its early stages. Many years may elapse before well marked clinical manifestations are obvious, so that it may not be recognized until old age is well established.

The exact *etiology* of portal cirrhosis is still unsettled. For many years, from a clinical standpoint, alcohol has been regarded as an important causative agent. On the other hand, cirrhosis is frequently observed in those who have never used alcohol. The condition is not infrequently found in individuals of races and sects to whom alcohol is forbidden. Recent experimental evidence has shown that experimental cirrhosis analogous to the portal cirrhosis of man can be produced by the prolonged use of a protein deficient diet or by diets that are lacking in vitamin B complex.^{20, 21} It is impossible to produce cirrhosis in animals by alcohol alone. Alcohol will only cause cirrhosis when its ingestion is combined with protein or vitamin B deficiency or when some additional toxic substance, as a chlorinated hydrocarbon, is used in conjunction with it.²² There is good reason to believe that alcohol is not the primary cause but rather an additional factor which, when associated with a chronic dietary and vitamin B complex deficiency, is capable of producing portal cirrhosis. It has been observed that even consistent drinkers who eat adequate diets avoid cirrhosis. Other toxic substances besides alcohol are probably at times contributory factors in portal cirrhosis. What effect the recently observed epidemics of infectious hepatitis and homologous serum jaundice may have upon the occurrence of portal cirrhosis remains for the future to determine.

Symptoms The symptoms of portal cirrhosis are essentially those of portal obstruction. Later, as the liver parenchyma becomes more involved, evidences of toxemia supervene due to hepatic insufficiency. Because of compensatory anastomoses that exist between the portal vein and the systemic circulation, the signs of portal obstruction may be delayed for a considerable period of time.

The early symptoms are usually referable to the gastrointestinal tract and are those of chronic *gastritis*, due to the chronic passive congestion of the stomach. Soon patients complain of fatigue. Loss of weight becomes a conspicuous symptom because of their inability to eat properly.

The most important obstructive phenomenon is *hematemesis*, the result of varices which develop under the mucous membrane of the esophagus because of the anastomoses between the coronary vein of the stomach and the esophageal veins (see p. 516). The vomiting of blood may be the first symptom to call attention to the presence of portal cirrhosis. To a greater or less extent it occurs in most patients at some time during the course of the disease. At times it may prove fatal.

Ascites present in well over 50 per cent of cases, is usually an obtrusive symptom of the disease. The disease is usually well established before ascites appears, although occasionally its onset may be rapid. It, too, may be the first symptom which calls attention to the existence of a cirrhotic liver. The ascites is not entirely due to intrahepatic compression of the portal vein. Thrombosis of the vein or some of its main branches is an added factor, in many cases it is due to the development of peritoneal irritation. The investigations of Tumen and Bockus²³ show that a hypoproteinemia, due to a decrease in serum albumin, is usually present in portal cirrhosis. This alteration in the blood osmotic pressure may well be an important factor in the production of ascites. According to recent studies by Hoffman and Lisa²⁴ portal hypertension has a greater influence on the production of ascites than does hypoproteinemia. The occurrence of cardiac failure later in the disease may also contribute to the abdominal effusion. Ascites may develop while the liver is still enlarged or the enlarged liver may exist without ascites. It is rarely absent when the organ reaches the true atrophic stage, however.

The liver is enlarged in the early stages, and sometimes throughout the whole course of the disease. As pointed out by Kelly,²⁵ the size of the liver varies greatly in different cases and in the same case at different times. Later in the disease, when the liver becomes atrophic, it may be markedly diminished in size. In most cases the size of the organ diminishes with advancing age.

By the time the liver shows definite changes the *spleen* has enlarged from congestion resulting from obstruction of the splenic vein, and has become palpable.

Dilated veins may appear on the abdominal wall and the lower chest wall due to efforts to establish compensatory anastomoses. These veins may become particularly prominent about the umbilicus, giving rise to the so-called caput Medusae which, however, is never so conspicuous in portal cirrhosis as it is in obstructions of the inferior vena cava.

Anemia becomes marked in the later stages of portal cirrhosis. Often this anemia is of a macrocytic type, closely resembling pernicious anemia, suggesting that the formation or storage of the hematopoietic substance is interfered with in hepatic disease of this type. The skin becomes pale and sallow. The face presents a typical pinched appearance with dilated venules over the cheeks. Crops of small angiomas scattered over the skin begin to make their appearance. There is a decided weight loss and asthenia develops. Bleeding from various mucous membranes may occur, notably from the gums, the intestines, the hemorrhoidal area, and the nose. Petechiae may appear in the skin and mucous membranes.

Jaundice is not a prominent symptom of portal cirrhosis. It was noted in one half of the author's series of cases.²⁶ It is usually of short duration, variable and slight.

Impaired liver function and disturbed nutrition sooner or later bring about manifestations of *intoxication*. The milder ones, such as headache, drowsiness and depression, may give place in the later stages of the disease to such serious symptoms as delirium, convulsions, paralyzes, and coma.

Terminal infections, due to the lowered resistance of the cirrhotic patient to bacterial invasion, frequently complicate the disease.

Diagnosis. In well developed cases presenting the classical symptoms, the diagnosis is usually not difficult. The early diagnosis which is so essential, if constructive efforts at treatment are to be instituted, is often exceedingly difficult because the early symptoms are vague and indefinite. In old individuals the *differential diagnosis* must be made from other conditions which may be characterized by *gastrointestinal hemorrhage*, such as *gastric and duodenal ulcers*, *gastric carcinoma*, and a chronic form of *splenic anemia*. The ascites of portal cirrhosis must be differentiated from that due to a failing heart, chronic forms of *peritonitis* or malignancy of the peritoneum. The large liver of portal cirrhosis may readily be confused with that due to carcinoma of the liver, amyloid disease, fatty infiltration, and chronic passive congestion, all of which may be found in the aged.

Among the *tests* indicative of impaired liver function that are of value in the diagnosis of portal cirrhosis may be mentioned the estimation of the serum bilirubin, the retention of bromsulfalein, and an increase in the urobilinogen in the urine, which is one of the most delicate tests indicative of parenchymal liver damage. Other useful tests are hypoproteinemia with reversal of the serum albumin globulin ratio, alimentary galactosuria, and the cephalin-cholesterol flocculation test. Peritoneoscopy will often prove to be a great diagnostic aid, especially when combined with a biopsy of the liver. It should be employed when the diagnosis is in doubt.

The ultimate *prognosis* of portal cirrhosis is bad. The disease may, however, run a prolonged course and symptoms may be latent for years. It is rare for more than three years to intervene between the advent of recognizable symptoms and death.

Treatment. If treatment is to be successful it should be instituted early before the liver changes are irreversible. Hence the importance of prompt diagnosis. The first step in treatment is to remove the underlying toxic cause, if that can be determined. To this end *all forms of alcohol* should be prohibited absolutely. All recent observers are in agreement that proper regulation of the diet is essential. The diet should be high in carbohydrates, high in protein, low in fat, and supplemented by brewers' yeast or vitamin B complex. Patek²⁷ advocates a diet which contains 3691 calories made up of protein 139, fat 175, carbohydrate 365, which includes 25 gm. of powdered brewers' yeast. If the latter is poorly borne vitamin B complex by mouth may be substituted. In addition thiamine chloride (5 mg.) is injected daily and liver extract in 5 cc. doses twice a week. The tendency to constipation should be overcome, but excessive purgation is undesirable. Should the Wassermann reaction be positive, antisyphilitic treatment in the form of iodides should be administered. It is a useful rule to administer iodides in all cases of enlarged liver of unknown

or obscure etiology. More than one suspected carcinoma or cirrhosis of the liver has disappeared under such treatment.

Hematemesis calls for complete rest in bed, with nothing by mouth for several days until the bleeding has ceased. When the tendency to bleed persists vitamin K has proved useful. The thirst may be allayed by salt solution per rectum. If there is much restlessness, morphine is indicated and if the bleeding has been severe, small transfusions may be necessary.

As a rule, the most obtrusive and troublesome symptom is *ascites*. Every thing should be done for the relief of this symptom, to delay as long as possible the inevitable paracentesis of the abdomen. The diet should be low in salt, not necessarily salt free, and the fluid intake should not be over 2000 cc daily. Active purgation and diuresis may aid in eliminating the excessive fluid. When there is no serious gastrointestinal irritation or renal inflammation the mercurial diuretics, such as novasurol, salyrgan, or mercupurin, have all proved useful. The effect of these mercurial diuretics is greatly enhanced by giving preliminary courses of ammonium chloride, preferably 10 gm by mouth, daily for several days. If and when these measures fail, resort will have to be had to paracentesis. It should be repeated whenever the abdominal distention interferes too much with the comfort of the patient.

The more radical surgical procedures that have been advocated from time to time have proved disappointing and should not be undertaken lightly in old people.

Biliary Cirrhosis. There are other forms of cirrhosis of the liver which may, at times, be met in old people, although their incidence is much greater in those younger than fifty. These include the forms of chronic inflammation of the liver that have been referred to as biliary cirrhosis because the outstanding clinical symptom is jaundice and the basic pathologic process involves the

which may be hematogenous or due to an ascending infection through the bile passages, is responsible for damage to the liver. In others mechanical obstruction to the flow of bile is the cause.

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the biliary tract. Gallstones are the commonest cause for such an obstruction but it may occur as a result of carcinoma of the head of the pancreas, displaced glands, aneurysms, adhesions, and other obstructive conditions. Since these causes of obstruction are not uncommon in old people, they may suffer from obstruction. In most cases infection becomes a factor. There is both clinical and experimental evidence that biliary obstruction and infection may coexist for any length of time. Fibrosis occurs, so that ultimately a certain amount of cirrhosis develops.

The *symptoms* of obstructive biliary cirrhosis are at first those of the underlying conditions. Symptoms of chronic jaundice are added to these later on. Most cases give a history of recurring attacks of temporary, incomplete biliary obstruction. With each succeeding attack, the jaundice becomes more severe. Finally, as the obstruction becomes more permanent, the jaundice persists and deepens, liver damage occurs, and symptoms of a toxic state supervene. When obstruction is intermittent and infection coexists, it is not unusual to have recurring attacks of chills, fever, jaundice, prostration, and leukocytosis, all of which subside partially or completely as the obstruction temporarily lessens. Such a clinical picture has been referred to as Charcot's intermittent biliary fever. In this form of biliary cirrhosis, the liver is enlarged and smooth and shows variable degrees of tenderness.

Biliary cirrhosis must be differentiated from those forms of liver disease in which the enlargement of the liver and the jaundice are due to primary disease of the liver cells themselves, rather than to a biliary obstruction.

The essential feature of the *treatment* is to determine the cause of the obstruction and remove it, if this is possible. If the biliary obstruction can be removed before the liver cells have undergone too much damage, complete recovery may ensue. When this is impossible, the ultimate outlook is bad.

Chronic Perihepatitis. Although not strictly one of the cirrhoses, the condition known as capsular cirrhosis, a better term for which is chronic perihepatitis, may properly be considered with the cirrhoses. In this condition, the enveloping capsule of the liver becomes markedly thickened because dense layers of hyaline connective tissue, which may attain a thickness of several millimeters, have been laid down. The liver looks as if it were frosted, hence the term "sugar icing" liver or "zuckergussleber." The disease usually occurs between the second and the fifth decades of life. The etiology is obscure. It probably results from the effects of bacterial infections or their toxins. In some, such as those who have suffered from rheumatic fever, the condition may be associated with chronic adhesive pericarditis and inflammation of other serous cavities. Under such circumstances it forms part of the picture of multiple serositis or Pick's disease. In old people it may occur without any demonstrable pericarditis and is said to be associated with arteriosclerosis and chronic nephritis. The liver may become distorted and compressed as a result of the capsular thickening. The spleen may become involved in the same process and frequently enlarges. Intrahepatic fibrosis is inconspicuous. Rarely does the liver parenchyma seem to be damaged.

The *symptoms* of this condition are indefinite. Frequently it is discovered accidentally. The existence of a chronic pericarditis and other chronic inflammations of serous membranes often attracts attention to the possibility of such perihepatic involvement. In some cases the most important symptom is ascites, probably due to portal obstruction or chronic irritation of the peritoneum or both. When ascites occurs, it is frequently excessive and tends to recur rapidly after paracentesis.

The condition must be differentiated from portal cirrhosis. In chronic perihepatitis, the course is more prolonged, the general condition of the patient suffers less, evidences of liver insufficiency are lacking, jaundice is absent, and the coexistence of chronic inflammation of serous cavities, particularly the pericardium, is much more common.

TUMORS OF THE LIVER

The only important tumors of the liver observed in old age are those which are malignant. Even these are infrequent in the old and are rarely encountered after the age of sixty. Such malignant tumors may be primary or secondary and are either carcinomata or sarcomata.

Carcinoma. *Primary carcinoma* may spring from the liver cells themselves as a hepatoma or may originate in the small bile duct as a cholangioma. According to Eggel primary carcinoma of the liver is found once in every 2000 autopsies, an incidence of 0.05 per cent. It may be single and massive or multiple and nodular. The right lobe is involved more frequently than the left and males slightly more often than females.

The *secondary form* is at least twenty-five times more common than the primary and is more frequent in women than in men. Secondary growths are multiple metastases from the stomach, esophagus, pancreas, gallbladder, cecum, sigmoid, breast, and uterus. Secondary involvement of the liver may occur, however, from any primary carcinoma in almost any part of the body. Some involvement of the liver is noted in about 50 per cent of all persons who die of carcinoma.

The most conspicuous *symptom* is enlargement of the liver, which is progressive. The liver may attain considerable size. It is nodular, hard, and sometimes tender. Whether symptoms of portal obstruction such as ascites, an enlarged spleen, dilated veins, and hemorrhages of the mucous membranes occur, depends entirely upon the position of the tumor mass. The same may be said in reference to jaundice. The symptoms that are common to all malignant growths of the liver are digestive disturbances, progressive loss of weight and strength, development of a marked anemia, varying degrees of pain in the upper right abdomen, irregular fever, and frequently a moderate leukocytosis.

The *diagnosis* may at times be difficult but is usually made by noting the progressively enlarging liver which is nodular, the cachexia, and the presence of or the history of a primary malignant growth elsewhere.

Sarcoma. Sarcoma of the liver is a rare disease at best. It may be primary or secondary and occurs chiefly in early life although occasionally rare cases have been reported in old people. The variety most likely met with in later years of life is a secondary melanotic sarcoma. The clinical course of sarcoma differs in no way essentially from carcinoma except that it runs a much more rapidly fatal course.

BILIARY TRACT

PHYSIOLOGICAL CONSIDERATIONS

The frequency with which disease of the gallbladder, with or without gallstones, is encountered in old people makes this group of conditions of unusual clinical importance and justifies a consideration of what little is known of the way in which age *per se* modifies the physiology of the gallbladder. Ivy²⁸ points out that the most important functions of the gallbladder are to concentrate hepatic bile and to evacuate it. He states that there are no reliable data on the effect of age upon the ability of the gallbladder to evacuate, although it is well recognized that this function is markedly altered by diseased conditions. Boyden and Grantham²⁹ studied the effects of age on the evacuation of the gallbladder and came to the conclusion that from childhood

to old age the rate of emptying of the female gallbladder is fairly constant, whereas in males at puberty something happens to slow down the rate of evacuation, from then until old age, the gallbladder of the male empties more slowly than that of the female. Their observations were carried out upon a group of older people with apparently normal gallbladders so that whatever alterations were noted in their older subjects were evidently due to age itself. From their observations one may justifiably conclude that there is no conspicuous lessening of the ability of the gallbladder to evacuate as the result of age alone, for they found that the gallbladder of a young child emptied more rapidly than that of the young adult and that the evacuation time of the gallbladder of the latter was more prolonged than that of older persons. The same observers came to the conclusion that the size of the gallbladder bears no significant relationship to its emptying time.

Although it was long believed by some that the gallbladder of old people atrophied, there is evidence to suggest that the contrary is the case, and that in old age the musculature in the walls of the gallbladder may undergo some hypertrophy and at the same time develop a tendency to sag the result of lost elasticity and possibly absorption of upper abdominal fat. Goodpasture³⁰ found the mucosa of the gallbladder in old dogs to be definitely thickened.

Sex is an important determining factor in gallbladder disease. There is an abundance of clinical evidence to show that all varieties of such disease are much more common in women than in men, particularly in women past the age of forty. Since, as above indicated, the gallbladder in a nonpregnant adult female empties more rapidly than that of the adult male, this increased frequency of gallbladder disease in women must be due to something other than motor insufficiency of the gallbladder. According to Ivy³ the increased incidence of disease of the female gallbladder is due to some metabolic disturbance, which alters the composition of bile, and to the frequency of infection or stasis due to a disturbance of the sphincter of Oddi mechanism. Probably a combination of one or more of these factors is responsible. He²⁸ has shown also that the gallbladders of pregnant women empty much more slowly than those of normal females and that they are subjected to greater distention. Gerdes and Boyden³¹ demonstrated definitely that during pregnancy the gallbladder empties more slowly and more incompletely than was the case before pregnancy or in women who had never borne children. These observations explain the long recognized fact that gallbladder disease is particularly prevalent in women who have been pregnant.

CHOLECYSTITIS AND GALLSTONES

Incidence Cholelithiasis and its associated cholecystitis, with their com

about 25 per cent of autopsies, although their existence may have been unsuspected during life, also the frequency with which gallstones occur bears a direct relationship to advancing age and sex. Mueller Deham and Rabson³² cite a series of 1000 consecutive autopsies in which gallstones were found in 9 per cent between the ages of thirty and forty years, after seventy years they occurred in more than 50 per cent. In the same series, gallstones were seven times more common in women than in men during earlier adult life but this

incidence became nearly equal with advancing years. In this same group, the frequency of all forms of gallbladder disease increased from 20 per cent in the lower age brackets to 70 per cent in those who were old. In about 50 per cent of those who had abnormal gallbladders, the condition found was some degree of cholecystitis.

The tables published by Dublin and Lotka³³ show an incidence of gallstones in white males aged 55 to 64, of 6.5 per cent, for white females during the same age period, 22.4 per cent. Between the ages of 65 and 74 the incidence for white males rose to 10.6 per cent and for white females to 30 per cent. During the same age period, the incidence among Negroes was definitely less; between the ages of 55 and 64 it was 3 per cent for males and 7.2 per cent for females, in the older age period, 3.9 per cent for males and 10.9 per cent for women. Jaffe,³⁴ in a statistical study of cholelithiasis based upon 2621 autopsies, found gallstones in 6.75 per cent of white males aged 51 to 60 and in 17 per cent of females. Between the ages of 61 to 70, the percentage in white males rose to 11.2 per cent and in females to 29.03 per cent. In those older than 70, the percentage in white males was 13.9 per cent and in females, 24.44 per cent. In these statistics also, the incidence in the Negro was definitely lower. In Negro males between the ages of 61 and 70, incidence was 4.34 per cent, for Negro females between the ages of 61 and 70, 10.9 per cent.

fourth decade, the incidence is definitely greater in women, in both sexes the frequency increases with age, reaching its maximum in the sixth to seventh decade.

Etiology. There is still lack of complete agreement as to the exact mechanism responsible for the production of gallstones. The three principal factors are *infection*, *stasis*, and *hypercholesterolemia*. These causes may operate singly or in combination, since it is unusual to encounter a cholelithiasis in which one or more of these factors does not exist or has not been active at some time. Evidence indicates that in most instances infection of some kind plays a leading role. This is particularly true of the mixed stones, a common variety made up of bilirubin calcium with some cholesterol, and of the rarer pure bilirubin calcium stones. The presence of bacteria, particularly of the colon bacillus-typhoid group, in the nidus of gallstones is added evidence in favor of infection. On the other hand, the pure cholesterol stone is frequently met in some condition such as pregnancy and in other instances in which no infection can be demonstrated. Impaired biliary drainage is undoubtedly a contributory factor in many cases. Gallstones may occur even when there is no demonstrable evidence of stasis. Infections and inflammations of the upper gastrointestinal tract are held to be an important etiologic factor. Among other contributory factors may be mentioned obesity, sedentary habits, diets rich in cholesterol, alcoholism (because of its effect upon the mucous membrane of the intestinal tract), ptoses, and displacements of the liver and gallbladder, as well as pregnancy and lactation. Whatever the factors responsible in calculus production, it is undeniable that their operation becomes more effective as age advances. Particularly is this true of women.

Symptoms. It is common clinical experience that the symptoms of gallstones and chronic gallbladder disease are less clear-cut and more indefinite

in old people than in younger people. As is well shown by autopsy, operative and roentgen-ray statistics, there are many older people who go through life harboring gallstones without presenting any symptoms that can be ascribed to gallbladder disease. In these, the cholelithiasis may be discovered accidentally, by operation for some other cause or in a routine x-ray examination. The existence of *symptomless gallstones* in older people is doubtless much more common than is usually believed. Such patients may complain from time to time of upper abdominal distress without definite attacks of gallstone colic. Their chief complaints and most persistent symptoms are referable to the stomach and consist of such disturbances as loss of appetite, nausea, epigastric distress, fullness and upper abdominal distention, excessive belching and eructations (see p 517). In such cases the true cause of the digestive disturbance may be overlooked, unless the possibility of gallstones is kept in mind and the diagnosis established by cholecystography.

In another group of older persons, and perhaps in the majority, a chronically inflamed gallbladder, with its gallstones, presents the well recognized symptoms of that condition. Such cases are characterized by the recurring attacks of typical upper abdominal or epigastric pain, characteristically referred to the region of the right shoulder blade. Tenderness in the right hypochondrium usually occurs with the attack. If much inflammation of the gallbladder exists, this is associated with right upper abdominal rigidity, fever, and leukocytosis. The gallbladder may be palpable, varying degrees of transient jaundice are noticed in most cases, and there is apt to be anorexia, nausea, vomiting, and associated gastric symptoms.

The group presenting the most difficult diagnostic problems comprises those in whom severe disease of the biliary tract develops with clinical manifestations so vague and insignificant that their true nature goes unsuspected until a severe abdominal catastrophe occurs. This possibility must always be thought of when dealing with old people with upper abdominal complaints. It is one of the peculiarities of old age that the most severe form of inflammatory disease of the abdomen may exist with a minimum of pain, with little or no constitutional reaction, and with local signs so slight that they may be overlooked easily (see p 537). As an example of this, we have observed gangrene of the gallbladder which has come on with great rapidity, has been accompanied with little or no pain, tenderness, rigidity, or other signs of peritoneal inflammation. Numerous instances are on record of rupture of the gallbladder occurring in old persons with gallstones and cholecystitis which were unrecognized and unsuspected, even when surgical intervention was resorted to as an exploratory procedure. Not infrequently cases of chronic cholecystitis have been regarded as so slight that surgical interference has not even been suggested, yet the patients were later found to have been suffering from slow perforation of the gallbladder with the subsequent development of an inflammatory mass in the abdomen, due to pericholecystitis and the establishment of a fistulous tract between the gallbladder and adjacent viscera. Such patients have recovered without operation, only to have the condition discovered when obstructive adhesions of the upper abdomen have developed.

Diagnosis. The diagnosis of gallbladder disease in old persons offers little difficulty if the case presents the classic symptoms of gallstone colic. The chief difficulty arises in the recognition of the atypical and latent cases in which gallbladder symptomatology is negligible. As has already been suggested, to rec-

ognize this group one must constantly bear in mind the frequency with which gallstones and chronic gallbladder inflammation occur in older people and then insist upon a thorough, routine gastrointestinal examination which should include a proper x-ray study of the biliary tract. With the modern diagnostic methods that are available, it is usually possible to reach a definite conclusion in cases of this type. It is in this group that transduodenal biliary drainage offers definite diagnostic possibilities. When properly carried out, the finding of bilirubin calcium crystals and bile pigment in the B bile is of definite diagnostic value. This has been emphasized by Bockus and Tumen, who found that in cases of suspected gallstone disease, such findings compared favorably with those obtained by cholecystography.

In old people, as in younger, when gallstones manifest themselves by upper abdominal pain and signs pointing to the right hypochondrium, the condition has to be differentiated from gastric and duodenal ulcers, pyloric spasms, obstructions high up in the intestines, pancreatic disease, appendicitis with a high, posteriorly placed appendix, and disease of the right kidney.

Differential Diagnosis. *Disease of the coronary arteries* is of equal importance and often more difficult to differentiate from gallstones, particularly in older people with arteriosclerosis and myocardial damage. This is particularly true when gallstones obstruct the cystic duct and the pain is characteristically epigastric, for not infrequently in occlusion of the posterior branches of the coronary arteries, the pain is not so much precordial as subxyphoid and epigastric. The relationship between disease of the gallbladder and coronary artery disturbances has been the subject of considerable investigation in recent years and should never be lost sight of in dealing with gallbladder disease in older people, in whom the coexistence of the two conditions is not infrequent. Differentiation between these conditions is further complicated by the fact that fever, leukocytosis, and upper abdominal tenderness may occur in both. A careful history eliciting symptoms of preceding cardiac disease on the one hand, or symptoms suggestive of biliary disease on the other, is undoubtedly helpful. In many instances, however, a final diagnosis cannot be arrived at without a cholecystogram and an electrocardiographic study (see p. 399).

When a gallstone enters the common bile duct it may cause complete obstruction, thus increasing the diagnostic difficulties markedly. It is particularly difficult to determine the cause of *persistent jaundice* when, as not infrequently happens, the obstruction of the common bile duct has not been accompanied or preceded by attacks of pain. The first problem is to decide whether the jaundice is of obstructive origin or whether it is due to diffuse hepatocellular disease. As has already been suggested, the latter is less common in old people than in younger individuals. On the other hand, it must be considered. In addition to a careful history and possibly a transduodenal biliary drainage for diagnostic purposes, the diagnosis depends chiefly upon the proper carrying out and interpretation of the various tests that have been shown to be useful in differentiating obstructive from nonobstructive jaundice. In obstructive jaundice, bile is usually present in the urine in greater quantities in proportion to the serum bilirubin level than are found in the nonobstructive variety. When the obstruction is complete, urobilinogen is not found in the urine, whereas variable quantities usually are present in the urine if the icterus is nonobstructive. The blood cholesterol is increased in obstructive jaundice and the percentage of cholesterol esters is normal, whereas in the nonobstructive type the

percentage of cholesterol esters is decreased. The galactose test is one of the most useful. It is usually positive if performed within two weeks of onset in nonobstructive jaundice, but is negative in the obstructive variety. Blood phosphatase is normal in nonobstructive jaundice and is definitely increased when actual obstruction exists. Hippuric acid synthesis shows a marked decrease in the nonobstructive variety and is normal in obstructive jaundice.

The type of obstructive jaundice most apt to be confused with that due to gallstones obstruction results from *carcinoma of the head of the pancreas*. This is one of the commonest and, at the same time, one of the most important differential diagnoses to make, particularly as both conditions are more apt to occur in old people. When gallstone obstruction is preceded by pain, the diagnosis offers less difficulty. When, however, individuals with chronic jaundice due to obstruction give no history of pain, the problem becomes more complicated. Courvoisier's law, namely, that in chronic jaundice due to gallstones the gallbladder is not palpable, but when the gallbladder is enlarged the obstruction is probably due to something outside the biliary tract, is by no means infallible, although it is helpful. Carcinoma of the head of the pancreas is apt to present a more severe and progressive weight loss and anemia. In many cases positive differentiation can be made only by exploratory laparotomy, a justifiable procedure unless the general condition of the patient is extremely bad, for if the obstruction is due to gallstones it can be removed and complete recovery may ensue, and, even if the obstruction is found to be due to carcinoma of the head of the pancreas, palliative surgery will at least relieve the jaundice and its attendant troublesome symptoms. If there is any question the patient should be given the benefit of the doubt. This was well exemplified in a case which came under our observation. A man in his late sixties who complained of loss of weight, fatigue, and anemia developed a chronic, painless jaundice. The gallbladder was not palpable. There was no history suggestive of gallstones. A diagnosis of carcinoma of the pancreas was made, at operation, the pancreas was found normal, but a large solitary gallstone blocked the common duct. Its removal was followed by complete recovery of the patient, who thereafter enjoyed vigorous health for a decade. He finally succumbed to an acute infection of the respiratory tract.

Complications. Gallstones may be responsible for various complications. The gallbladder may become acutely inflamed and rupture, or perforate. If gallstones completely occlude the cystic duct, the gallbladder becomes markedly distended with mucoid secretions which are no longer able to drain out through the cystic duct and a condition of hydrops of the gallbladder results. Complete obstruction of the common bile duct, if not relieved, may bring about secondary disturbances of the liver which have already been referred to under obstructive biliary cirrhosis. Gallstones may perforate either the gallbladder or any of the ducts in which they become lodged and may ulcerate into the small intestine or into the peritoneum. Such a process is accompanied by chronic peritoneal inflammation. A gallstone may lodge at the lower end of the common bile duct where it enters the duodenum, bringing about a partial intermittent obstruction. When this is associated with infection, patients suffer from recurring attacks of chills, fever, sweats, and varying degrees of jaundice. Gallstones may be responsible for a chronic pancreatitis and in some instances they may bring about acute hemorrhagic pancreatitis. Carcinoma of the pancreas has been definitely shown to be more frequent in

persons who have gallstones than in those who have not. The same statement applies to carcinoma of the gallbladder. These are two of the later and more serious complications of chronic cholelithiasis which furnish an important reason for the removal of gallstones, even when they are causing no symptoms.

Prognosis The prognosis of chronic gallbladder disease due to gallstones depends entirely upon two factors: first, whether the gallstones remain quiescent in the gallbladder, or whether, in attempting to leave the gallbladder, they bring about some of the above mentioned obstructive phenomena, and second, the presence of infection. When neither of these circumstances supervenes, the patient may live in comfort for many years, even with a gallbladder crowded with stones.

Treatment The treatment of chronic gallbladder disease and gallstones in the aged requires the physician's best judgment. In young people when a diagnosis of gallstones has been made the obvious and safe procedure is to advocate their removal by a competent surgeon. In older people, however, much more conservatism must be shown and the entire condition of the patient must be carefully evaluated before he or she is urged to seek operation. This is particularly true of those in whom gallstones exist but who are practically symptom free. In such, particularly if their cardiovascular apparatus shows evidences of age, it is often the part of wisdom to avoid operation unless some of the urgent complications of cholelithiasis ensue. It is, therefore, both unwise and unnecessary to insist routinely upon operations for gallstones in all old people. The indications for operation should be definite and the general condition of the individual sufficiently good to eliminate any undue operative risks. Numerous instances could be cited of old people, living in comparative comfort and safety in spite of their gallstones, who submitted to an operation, theoretically indicated but practically unwise, only to succumb promptly to some of the postoperative complications so frequently encountered in older individuals.

Carefully considered *medical management* will afford considerable relief to the majority of older people who are suffering from uncomplicated biliary tract disease. A careful appraisal should be made of their entire physical condition. Measures should be instituted to put their nervous systems, their cardiovascular apparatus, and their kidneys under as little strain as possible. In prescribing a *diet* their condition of nutrition should be taken into consideration. Theoretically, the diet should be low in fats, high in carbohydrates but of adequate caloric value, and one which supplies sufficient vitamins. It is difficult to alter and to regulate the dietetic habits of many old people, who frequently do better when given some latitude and allowed to follow that form of diet which is most agreeable to them rather than attempting to force them suddenly to adopt a distasteful dietetic program. In old people particularly violent purgation should be avoided, rather their bowels should be moved regularly and thoroughly by mild laxatives. Here again the lifelong habits of the individual must be given due consideration. Various preparations of bile salts enjoy a reputation in the treatment of chronic gallbladder disease and doubtless in some instances have proved of advantage, but should be avoided if evidence of parenchymal liver damage exists. Biliary colic requires the use of rest, local heat, and the hypodermic administration of nitroglycerine or morphine and atropine. In the treatment of gallbladder disease of the aged,

the watchword should be conservatism. Radical procedures should be avoided unless imperatively indicated and every effort should be made to disturb as little as possible the environmental factors and life habits of the older patient.

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SECTION V

DISORDERS OF THE GENITOURINARY SYSTEM

CHAPTER 37

THE NEPHROPATHIES

EDWARD J. STIEGLITZ AND SERUCH T. KIMBLE

THE human kidney decreases in size and vascularity with advancing age even in the absence of specific renal disease, and it is indeed difficult to draw the line between normal and pathologic renal senescence. Alterations of renal structure are predominantly secondary to vascular change, a normal part of the aging process (See Chapter 4.) The old organ is macroscopically smaller, while microscopic examination reveals obliteration of many functional units by scar tissue and various degrees of compensatory hypertrophy of those units which remain. Modifications of function accompany those of structure, so that the old kidney is qualitatively as well as quantitatively different. The effect of these nephrosclerotic changes upon the physiology of the patient depends upon their extent and rate of progression, as well as concomitant disorders.

The kidney plays an important role in determining the character of the aging process. That nephropathy and hypertension are closely associated is common knowledge, although the exact mechanisms are not fully understood. Etiologic possibilities are as follows: (1) renal injury may produce hypertension, (2) hypertensive disease may injure the kidney by impairing its circulation, and therefore its nutrition, and (3) a common irritant may damage both arterioles and renal parenchyma.¹ Renal ischemia is the basic factor, and depending upon its permanence, degree, and rate of progression, the patient may develop the clinical picture of benign or malignant hypertension. It is notable that the working kidney utilizes more oxygen than any other organ in propor-

In approaching the problem of nephropathy in geriatric medicine, one must consider not only the peculiarities of the disease or diseases, and the physiologic consequences upon the individual as a whole, but also the physiologic characteristics of senescence and senility. Attention will be given first to these subjects and then focused upon their effects upon diagnosis and therapy.

CHARACTERISTICS OF AGING AND THE AGED PATIENT

Lowered Homeostatic Response. Physiologic constants, such as body temperature, blood sugar level, and pH of the blood, are not appreciably altered in the aged, but those mechanisms which maintain equilibrium or homeostasis under stress conditions are affected. Normal body temperature is the same at eighty years as it was at eight, but the octogenarian tolerates extremes of heat

and cold or sudden shifts in environmental temperature more poorly than he did in youth. A lower basal metabolic rate and diminished muscular activity contribute to a slower rate of heat production, while degeneration of sebaceous and sweat glands and poorer cutaneous vascular supply retard the rate of heat loss. The fasting blood sugar level is not significantly altered with age, but ability to cope with increases of glucose supply is diminished. Glucose tolerance tests produce a diabetic type curve accompanied by glycosuria in a far greater percentage of elderly individuals than in the young, indicating impairment of the capacity to store and utilize glucose.

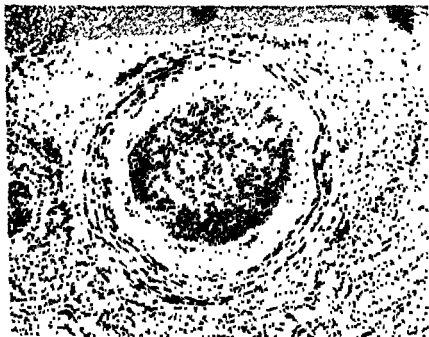
The hydrogen ion concentration of the blood is constant throughout life (pH 7.4), but as age advances there is a weakening of those mechanisms which maintain the acid base balance of the blood under stress conditions. Respiratory, cardiovascular, and renal factors are involved. The removal of carbon dioxide from the blood by the lungs is less efficient because of impaired pulmonary efficiency due to emphysema, weakness of the intercostal and accessory respiratory muscles, calcification of the costal cartilages, and kyphosis. The cardiovascular changes of senescence impede the removal of acid metabolites from the body tissues as well as interfering with their supply of oxygen. Increased vascular rigidity limits the adaptation of blood supply with fluctuating tissue needs. Thus a muscle supplied by a sclerotic vessel can receive but little more blood in the working than in the resting state. Local hypoxia and acidosis supervene. Cardiac factors further contribute to circulatory inefficiency, for even in the absence of specific cardiac disease the ventricles do not empty as completely as in youth, nor does the heart accelerate to the same degree under stress conditions. Therefore, the stroke volume and circulation rate vary over a relatively limited range. Finally, acid base balance is impaired by depreciations in kidney function. The healthy kidney conserves fixed base by excreting organic acid in the free state, producing ammonia, changing dibasic phosphate to the monobasic form, and forming hippuric acid. Unfortunately, as yet there are no clinical tests for measuring the renal reserves for these functions and thereby detecting their early impairment, but in frank renal decompensation, urinary ammonia is diminished, sodium chloride is wasted in the urine, while serum sulfate and phosphate concentration are elevated, and the serum bicarbonate content falls.

Previous Renal Injury. Obviously, the probability of previous renal injury increases with age. The silently accumulative injury and its functional consequences, however, are often difficult to detect. Disease in senescence is characteristically of multiple and cumulative etiology, which must be diligently searched for in the patient's past.³ The nephropathies are no exception to this rule.

Previous kidney damage may stem from a variety of causes. Repeated insults by bacterial toxins, particularly streptococcic and staphylococcic, and bacterial invasion from the blood stream or adjacent structures are not unusual. Mechanical obstruction in the lower urinary tract resulting in an ele-

episodes of renal ischemia cannot be denied. Actual renal damage is minimized, however, by the organ's lowered metabolic rate during the period of vasoconstriction,⁵ which mechanism is set off by either lowering renal arterial pressure or raising the interstitial pressure in the juxtaglomerular tissue. Injury to the tubules by arterial toxins as lead, arsenic, and mercury is mentioned but a few of the many causes of tubular damage. Lead, arsenic, and mercury, and also an arterial irritant, will produce a specific nephrosclerosis in individuals chronically exposed.

The effect of previous renal damage is not known in a patient with few



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reserve. He may be able to maintain his working load, but if he becomes febrile in

compensation may occur. The concepts of cardiac reserve and decompensation are familiar. In the case of renal reserve and decompensation

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acidotic bodies in the urine. Patients were unable to maintain a normal acid-base

equilibrium of the blood. Histologic study demonstrated that as animals advance in age there is a greater tendency for renal epithelial degeneration upon exposure to toxic substances. The observed alterations included edema, vacuolation, and an increase in stainable lipid, and were found primarily in the proximal and distal segments of the nephron. The vascular tissue did not show any increased susceptibility to the poisons in older animals. MacNider postulates that the toxic agents produced physical and chemical changes by inhibiting intracellular oxidation. He assumes that inhibition is more marked in older animals because the basic life processes are of a diminishing order quantitatively as they progress from youth to senility. This experimental work was performed upon dogs, but it seems logical to assume that similar factors operate in the case of man.

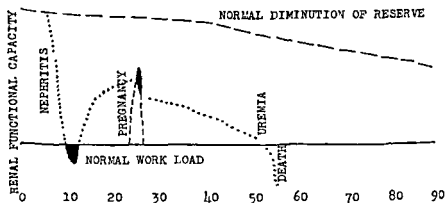


Fig. 121 At birth, renal functional capacity is roughly 400 per cent of the work load. Reserve capacity progressively diminishes with age, but under normal circumstances continues to exceed bodily requirements, which are relatively constant. In the case depicted above, an acute glomerulonephritis reduced the kidney reserve. When the latter fell below the work load, renal decompensation developed (dark area). Upon recovery, reserves were only partially restored. Pregnancy later increased renal functional requirements to the point where they exceeded the reserves and decompensation again supervened. At the termination of pregnancy, functional requirements resumed the normal level but kidney reserves were further curtailed. They depreciated at an accelerated rate, and the patient eventually died in uremia at age 55. This illustrates the concept of margin of safety, which may be depleted by several factors.

Lowered Extrarenal Reserve. In the evaluation and treatment of nephropathy in the aged, full cognizance must be taken of lowered extrarenal reserves, particularly those of the circulatory system (See Section V). The kidneys cannot clear the blood of metabolic debris unless the circulation is adequate. Diminution of circulatory reserves in senescence and senility is effected by both cardiac and vascular alterations. Coronary sclerosis reduces the availability of oxygen and glucose to the cardiac musculature, which, even

generative capacity is reduced and may fail in an emergency. Recovery from hemorrhage is slower, and the leukocytic response to infection is less marked (See Chapter 13.) Furthermore, as age advances there is a replacement of red by yellow marrow, the latter sometimes becoming gelatinous in wasting diseases.⁷ One may infer, then, that hematopoietic reserves are diminished in the aged, but further investigation of the problem is needed.

Even under normal circumstances the liver progressively decreases in weight after age sixty. Because of its huge factor of safety, clinical liver function tests, nevertheless, give normal values in healthy old age and demonstrable functional insufficiency does not develop in the absence of hepatic disease (See Chapter 36.) MacNider⁸ observed a diminished capacity of the liver to deal with toxic substances as age advances in dogs. In young animals, necrotic hepatic cells were normally replaced, while in old animals, replacement was made by an abnormal syncytial type of cell, which apparently functioned fairly well as a liver cell while acquiring an enhanced resistance to certain poisons upon reexposure.

Slower Rate of Tissue Repair. The rate of tissue healing is so closely correlated with age that DuNoüy⁹ was able to express the relationship mathematically. Knowing the subject's age and using DuNoüy's formula, one can predict fairly accurately the healing time of a clean wound and, conversely, given the rate of wound healing determine the subject's age. Generally speaking, wound repair requires an additional day for every additional five years of age, so the healing process is not only qualitatively different in the geriatric patient, but also proceeds at a slower rate.

CHARACTERISTICS OF NEPHROPATHY IN SENESCENCE AND SENILITY

Although they do occur, acute nephropathies are relatively infrequent after age fifty. As in younger groups, the incidence is greater among males and presents a steadily rising mortality rate with advancing age. In most instances the renal degenerative process is characterized by an insidious onset and a chronic, progressive course. Symptoms may be absent or deceptively mild.

Frequency of Concomitant Disease. The association of arteriosclerosis, hypertension, and nephropathy, being common knowledge, is mentioned only in passing. Anemia, malnutrition, and diabetes are also exceedingly common in elderly patients with kidney disease and materially influence therapy. Normocytic or macrocytic anemias are often discovered and are corrected with difficulty, being to some extent refractory to iron and liver medication. They occur in many types of renal impairment, the common factor presumably being chronic nitrogen retention and consequent depression of bone marrow function.¹⁰ The exact identity of the substance or group of substances responsible for the intoxicative type of anemia is as yet unknown. Every effort should be made to restore the hemoglobin to normal levels, for no organ, including the kidney, can function efficiently with an inadequate oxygen supply. Transfusion may be resorted to if other therapeutic measures fail, but should be performed with caution because of the risk of tubular damage.

Clinical and subclinical malnutrition is frequent in elderly persons, being caused by faulty dietary habits, hypochlorhydria, disturbances of gastrointestinal motility, and metabolic disease. The term malnutrition encompasses both hyper- and hyponutrition, but in the present discussion its use is limited to the latter sense. Hyponutrition may aggravate or be aggravated by renal

disease Anorexia, nausea, vomiting, and diarrhea accompany azotemia. Anorexia in the senile not infrequently is the major subjective symptom in the pre uremic state of poor renal compensation. The accompanying weight loss, pallor, and asthenia are too frequently attributed to the anorexia without exploration of the causes of appetite loss. In the presence of nephritic or nephrotic edema, the appetite may be good, but digestion and absorption of food are inefficient because of gastrointestinal edema. The patient's efforts in self medication and diets prescribed by the attending physician often further aggravate the hyponutritional state. Moreover, some of the diuretics commonly employed, for example, ammonium chloride and the mercurial diuretics, complicate the feeding problem by aggravating anorexia and gastric discomfort.

Persistent losses of serum protein in the urine very readily induce a state of negative nitrogen balance, and it is desirable, therefore, to measure the dietary intake and urinary loss of nitrogen. A positive balance must be maintained *not only to prevent or correct edema and malnutrition, but to combat anemia*, since iron and liver extract alone will not suffice if protein for hemoglobin and erythrocyte formation is not available. Stress should be laid upon chronic alcoholism as a contributor to malnutrition. Alcohol itself is a poor food and its excessive consumption often leads to the irregular and insufficient intake of dietary essentials. Alcoholic gastritis deranges digestion and absorption, while subclinical hepatic cirrhosis embarrasses carbohydrate and protein metabolism as well as vitamin storage.

The syndrome of hypertension and albuminuria develops with distressing frequency in patients with diabetes, even though blood sugar level and glycosuria have been adequately controlled with insulin.¹¹ Renal signs and symptoms unfold on the average ten years after appearance of the diabetes.

consist of intercapillary glomerulosclerosis and generalized vascular degeneration.

phasized. It must be kept in mind constantly if diagnoses are to be made and treatment instituted before irreparable damage has occurred. Attention often is not attracted to the kidneys by symptoms until their vast reserves are depleted and decompensation supervenes. The more subtle depreciations of renal reserve may be accompanied by a normal urinalysis, stress tests being the sole method by which they can be detected.¹² Renal disease is sometimes fortuitously discovered by a routine blood pressure determination, ophthalmoscopic examination, or chemical analysis of the blood. Initial symptoms as well as signs may be referred to other systems of the body. For example, persistent headache or the sudden blindness of retinal hemorrhage may be the first subjective signal of danger. Only by performing thorough physical examinations and not being satisfied by routine urinalyses in periodic health inventories, but carefully investigating the renal reserve with stress tests of kidney function, will physicians diagnose nephropathy in the elderly while there is still an opportunity to salvage appreciable kidney function.

Progressive Tendency. Degenerative disorders are distinguished by their chronic course and progressive nature. This progressive tendency may stem from the etiologic factors which originally provoked the disease, or may be

attributed to secondary perpetuating factors. The illnesses of youth, on the other hand, are usually acute, leave little or no residual damage behind them, and have no progressive tendency (See Chapter 2)

DIAGNOSTIC CONSIDERATIONS

Renal Function Tests. The most valuable diagnostic aid for detecting early renal embarrassment is the *renal concentration test*. There are numerous ways of performing this test, but the simplest is that of Fishberg¹³

The patient receives no food or fluids after the usual supper until completion of the test. At 7 A.M. he empties his bladder, and this specimen is discarded. At 8, 9, and 10 A.M., separate urine specimens are collected and labeled with the hour of voiding. The specific gravity of the specimens is determined, the highest value being taken as a measure of the renal concentrating power. At least one specimen should reach or exceed a specific gravity of 1.025. Readings of 1.020 or lower reflect definite impairment of reserve, while in renal decompensation the maximum concentration often is in the neighborhood of 1.010.

What does the concentration test accomplish? As a stress test it increases the renal work load by diminishing the amount of solvent in which metabolic wastes can be excreted. The kilocalories of work performed by the kidneys in elaborating a normal urine (approximately 0.8 kilocalories) are least when the twenty-four-hour urine volume is 2.4 liters.¹⁴ At volumes greater or less than this, and particularly at volumes under 1 liter, renal work is markedly increased. Kidneys with adequate reserve respond to conditions of water deprivation as imposed by concentration tests by excreting more solute per unit volume of urine, thus raising the specific gravity. The failure of diseased kidneys to concentrate solutes is proportional to their functional inadequacy. They excrete fewer solids and consequently a more dilute urine.

The renal concentration test has numerous advantages. It requires little apparatus and can be performed quickly. Therefore, it can be made a simple routine office procedure. Besides being inexpensive and simple, the test is safe, involving no risk to the patient. It is based upon well established, sound physiologic principles and normal responses to it are relatively uniform. Finally, it is sensitive, revealing early depreciations of kidney reserve when the results of other tests are still normal.

Like every human contrivance, the renal concentration test has limitations. In the presence of subsiding edema or ascites, false low values are obtained, for the mobilization of the retained fluid induces urinary dilution. If, on the other hand, the urinary specific gravity is high in the presence of edema, one can rest assured that the kidneys are functionally adequate and the edema of extrarenal origin. False low values for specific gravity are obtained also if the patient is uncooperative and drinks fluid during the test period, if he has drunk excessive amounts of fluid just preceding the test, or if he has diabetes insipidus. Concentration tests are further limited in applicability by the fact that they do not reflect progression of renal degeneration after the specific gravity reaches 1.010. In such circumstances the urea clearance test is more informative. Finally, febrile and surgical patients often present serious contraindications to the withholding of fluids for sixteen hours, so that some other device must be employed to evaluate their renal status. However, as fever is associated with an occult edema, or retention of fluid by the tissues, the urine is concentrated more than normally. If single voided specimens of urine reveal specific gravities in excess of 1.025, it is unnecessary to conduct the more

formal renal concentration test until later when it is desirable to establish a base line for future comparisons

Schneeberg¹⁵ has outlined a method which eliminates false low values with the concentration test and makes its use safe in persons unable to tolerate long periods of dehydration. He has the patient empty his bladder and then injects 10 units of posterior pituitary substance subcutaneously. Three urine specimens are then collected at hourly intervals. A specific gravity of 1.022 or higher in any one specimen is considered normal. The test is absolutely contraindicated if the patient is pregnant, is hypersensitive to posterior pituitary extract, or has severe coronary arterial disease. Its use is inadvisable in the presence of angina pectoris, hypertensive encephalopathy, or epilepsy.

Renal dilution tests are being discarded because of their lack of sensitivity and difficulties in interpreting the observations. Though they are stress tests measuring the renal faculty of handling excess amounts of water, this function is rarely impaired except in acute and obvious conditions. The *urea concentration test* of McLean and de Wesselow is another stress inducing procedure but it is difficult to evaluate because of several variables which are hard to control. The *urea clearance test* is an accurate guide to the effectiveness of glomerular function. While it does not impose conditions of stress and cannot approach the sensitivity of concentration tests in revealing very early reductions of renal reserve,¹⁶ it excels them in detecting improvement or exacerbation of impairment after extensive kidney damage has occurred. Its contraindications are negligible. Other renal function tests are based upon the excretion of certain nontoxic dyes. The phenolsulfonthalein (PSP) test is the most widely used of this group and employed in conjunction with ureteral catheterization sheds light upon the comparative output of the two kidneys. This test is primarily a measure of tubular function and although not sensitive in its total two hour elimination, is sufficiently accurate as a fifteen minute test in the catheterized patient to be clinically valuable. Low values are caused not only by renal disease but also by circulatory insufficiency, edema, and metabolic quirks leading to destruction of the dye within the body. False high values are occasionally observed in hepatic disease.

Clinical renal function tests, in summary, offer the physician valuable information regarding the effectiveness of the kidneys in regulating water balance, eliminating and conserving organic and inorganic solutes, and clearing the blood of foreign substances. They aid in the diagnosis, evaluation, and localization of the disease process, and should be performed when kidney damage is known or suspected to have occurred as well as when an increased burden upon that organ is anticipated, as prior to elective surgical procedures. The techniques of the various clinical renal function tests as applied to elderly patients differ in no significant manner from the methods with younger patients. Their intelligent interpretation involves awareness of the changes which take place with aging.

INTERPRETATION OF SIGNS

Proteinuria. Nephropathy is characterized by the presence of protein in the urine, even though its appearance be transient and the quantity small.

twenty-four hours, but that this is reabsorbed by the tubules. Proteinuria may result, then, either from increased glomerular permeability or diminished tubular reabsorption. If due solely to the latter factor, it will never be profuse. Proteinuria following the intravenous administration of gelatin or accompanying severe hemoglobinuria is best explained as being due to accumulation of these substances in the tubular cells and consequent tubular inability to reabsorb the protein of the normal glomerular filtrate. The glomeruli in lipoid nephrosis appear normal microscopically, whereas tubular destruction is extensive. Proteinuria in these cases is explicable on the basis of the above concept or of ultramicroscopic alterations of the glomerular membrane.¹⁷

Proteinuria is not proof of nephropathy, nor is its severity a reliable guide to the extent of kidney damage. It may be massive in a nephrosis from which the patient later recovers with little residual damage, or slight in a terminal chronic glomerulonephritis. Proteinuria may occur in the absence of organic renal disease, so-called "functional" or orthostatic albuminuria being a case in point. The urine secreted while the patient is lying down is free of albumin, but resumption of an erect posture is followed by its reappearance. This syndrome is caused by venous stasis and consequent renal congestion. Aberrant renal vessels, kinking of the pedicle, and renal ptosis are not uncommon factors. Orthostatic albuminuria is frequent in adolescence and early youth, especially in underweight lordotic patients, but is rare in later years. Presumably it does not lead to kidney disease. One should remember that the proteinuria of genuine nephritis or nephrosis may be similarly influenced by postural change and should exercise caution in making a diagnosis of orthostatic albuminuria. The absence of hypertension, edema, and urinary casts and cellular elements simplifies the diagnostic problem to a considerable extent. Cardiac decompensation, pyrexia, protein hypersensitivity and multiple myeloma are also attended by proteinuria. The underlying pathologic physiology in congestive failure is venous stasis, which leads to renal engorgement and histoxia and increased glomerular permeability. Febrile states are accompanied by transient cloudy swelling of the tubules, which presumably interferes with their reabsorption of protein normally found in the glomerular filtrate.

Patients sensitive to certain dietary proteins develop albuminuria following their ingestion, the kidney seemingly being selectively permeable to foreign substances. Bence Jones protein, found frequently but not exclusively in multiple myeloma, easily penetrates the normal glomerular membrane because of its small molecular size and is disposed of in the urine. Specific kidney disease, then, is not a prerequisite for proteinuria which may well be a defense mechanism, even in nephritis. Serum protein combines with toxic molecules in an effort to render them innocuous and thus form a foreign protein which is selectively eliminated by the kidneys.¹⁸

Edema. It is unnecessary to dilate upon the roles of increased capillary permeability, plasma protein depletion, and sodium chloride retention in edema formation. The facts are well known. One cannot infer from them, however, that in nephropathies the degree of edema parallels the extent or severity of renal damage. Nor is it correct to assume that edema reflects renal inability to excrete water, the proper amount of water available for excretion simply may not reach the glomeruli. Edema, like proteinuria, often develops in the absence of kidney lesions. It results from interference with venous blood flow, lymphatic obstruction, inanition, certain vitamin deficiencies, and al-

lergic states. These etiologic possibilities should be considered in differential diagnosis and, even though the kidneys have been incriminated, ruled out as contributory factors

Edema formation should be regarded as a defense mechanism, by which the body removes toxic substances from the circulation and stores them in the serous cavities and interstitial spaces, where they can do little harm to vital structures. During the subsidence of edema, this material, plus accumulated cellular wastes, reenters the vascular system for excretion. At that time the patient is subjectively sicker than during the period of edema formation and he is more likely to develop untoward cardiac and cerebral disturbances



Fig 122 Photomicrograph of renal arteriole, magnified 95 times. Extensive nephritis and nephrosclerosis. (From Stieglitz, *Arterial Hypertension*, Paul B. Hoeber, Inc., New York.)

Arterial Hypertension. Hypertension accompanies all varieties of Bright's disease except the focal nephritides and the nephroses. Although the incidence and intensity of arterial hypertension bear no constant relation to the extent of the renal pathologic process, they are the most sensitive index of acute exacerbations and a fairly reliable guide to the rate of progression.

In acute diffuse glomerulonephritis the elevated arterial tension varies in severity and duration. It may return to a normal level and remain there, provided the case does not progress to the subacute and chronic phases, or it may be so marked as to precipitate congestive heart failure or a cerebral accident. A complete and permanent return to normal values is infrequent, however, in senescence, for the majority of cases either pass directly into the subacute stage or become reactivated after a quiescent period and then become sub-

acute As irreversible arteriolar changes occur, the systolic pressure is persistently elevated and the diastolic begins to rise Ophthalmoscopic findings then consist of arteriolar narrowing, venous engorgement and tortuosity, and perhaps retinal edema and hemorrhages, which are very common among diabetic nephritics Persistent hypertension ultimately leads to cardiac decompensation and cerebral accidents, which may prematurely dispatch the patient before fatal renal insufficiency develops These are discussed in detail in Chapter 30

Azotemia An increase of nitrogenous products in the blood is usually considered to indicate faulty kidney function, but such is not always the case For example, uric acid, the first component to become elevated in early kidney insufficiency, is increased in gout, leukemia, polyarthritis, and hypertension of nonrenal origin¹⁹ Urea, the next constituent to rise, is excessive in dehydration states caused by such conditions as diarrhea, vomiting, hemorrhage, diabetes mellitus, and Addison's disease Diseases leading to rapid breakdown of protein within the body and its discharge into the blood stream, for example pneumonia and empyema, will produce abnormally high concentrations of nonprotein nitrogen If, however, the azotemia is of proven renal origin, its fluctuations accurately reflect the severity of the renal functional inadequacy The presence and severity of nitrogen retention offer no reliable clue to the type of nephropathy present²⁰ Further investigation is needed before one can state which substance or group of substances produces the clinical syndrome of "uremia," or precisely identify the renal pathology by analysis of serum constituents

THERAPEUTIC CONSIDERATIONS

A stereotyped or routine system of treatment for nephropathy cannot be formulated Consecutive cases are no more identical than are the individuals in which they arise Therefore, individualization of treatment is necessary for maximum therapeutic effectiveness Inherited and acquired physical and mental characteristics influence therapeutic programs just as much as they do diagnosis

Correcting Etiologic Factors Most nephropathy seen in the second forty years of life has its origin in the inaccessible past Predisposing and provoking etiologic elements are usually not open to correction The occasional case of acute glomerulonephritis is an exception to this generality and in such cases painstaking care should be used in eradicating foci of infection and enhancing the patient's resistance Scarlet fever and rheumatic fever, though uncommon after forty, do occur Recent research indicates that some streptococci infections produce specific autoantibodies which, upon reacting with kidney tissue, give rise to glomerulonephritis²¹ This mechanism may account for the renal consequences of both scarlet fever and rheumatic fever If adequate treatment be instituted promptly, however, and absolute bed rest be enforced, complete clinical cure is possible Chemotherapy and surgery during quiescent stages are often of value Sulfonamides should be administered with caution, though, because of the possibility of crystallization within the tubules and further impairment of renal function Concomitant alkalization and forcing fluids will obviate this danger, but prolonged alkalization should be avoided, for according to Ziegler and Brice,²² the ability of kidneys to excrete solids and concentrate the urine is greater when the urine is acid or neutral than when

alkaline. It has also been demonstrated that protracted alkalinization of the urine causes renal damage.²³ Eradication of renal and extrarenal infection is of paramount importance in pyelitis and pyelonephritis. It should be coupled, however, particularly if the disease is recurrent, with search for mechanical obstruction in the lower urinary tract. (See Chapters 38 and 39.)

Treatment of Anemia The anemia of chronic nephropathy is characteristically refractory to treatment. The diet should contain an abundance of the "intrinsic factor," and iron, crude liver, and folic acid may be therapeutically effective, particularly if extrarenal factors contribute to the anemia. Whole blood transfusion may be used as a last resort or in emergency situations, but should be performed with caution. The correction of anemia is profoundly important, for no organ can function efficiently without a liberal oxygen supply. Deficiencies of hemoglobin, and consequently of oxygen transport, cause generalized histanoxia. Cardiac embarrassment and hypertension are often markedly alleviated simply by correcting a hemoglobin deficiency. The kidneys in chronic nephritis, operating under the double handicap of intrinsic disease and superimposed circulatory embarrassment, are particularly vulnerable to histanoxia. Correcting anemia, therefore, specifically spares the kidney in addition to promoting the general well-being of the patient.

Diet. In the absence of edema, fluids should be liberally supplied, particularly if the nonprotein nitrogen is elevated. Several factors are to be considered in determining the desirable level of fluid intake. First, renal work load is minimal when the twenty-four-hour urine volume is 2.4 liters and rapidly increases for volumes below this figure. Secondly, chronically diseased kidneys fail to concentrate the urine, and thus require more water (solvent) per gram of metabolic debris (solute) to be excreted. As renal disease progresses, the former must be augmented if the latter remains constant.

In the presence of edema, regulation of water intake is a more difficult problem. One must supply abundant fluid to the vascular system, which is usually dehydrated, without excessively increasing the degree of interstitial hydration. This can frequently be accomplished by eliminating salt from the diet while continuing the liberal administration of fluids. Intravenous administration of fluids may be resorted to if gastrointestinal disturbances preclude use of the oral route. They should be given with caution, for recent studies indicate that the danger of circulatory collapse being induced by the intravenous administration of electrolytes is greater in nephritic than in cardiac edema.²⁴ Intravenous fluids rapidly extravasate into the tissues, later reentering the circulation via the lymphatics to increase the circulating blood volume. This occurs quite rapidly in patients with nephritic edema, occasionally causing cardiac distress and pulmonary congestion. In cardiac edema, infusions of isotonic solutions of electrolytes are relatively well tolerated, since lymphatic impairment prevents the rapid return of fluids to the circulation.

The diseased kidney loses its ability to regulate not only water balance but body mineral balance as well. Consequently, effort must be directed toward reestablishing proper body mineral content, which will to a large measure restore water and acid base equilibria. Sodium chloride intake should be minimized during edema mobilization, since the sodium ion holds water in the tissues. Restriction is unnecessary in the absence of edema and indeed may lead to chloride deficiency, diminution of free base, or frank acidosis. Salt requirements are increased in nephropathy, for the tubules fail to conserve so-

dium and chloride ions, excreting them at a relatively constant rate, even in the presence of a serum deficit. Replacement therapy is obviously indicated.

Before deciding the amount of salt to be included in the diet, one should determine the plasma levels of bicarbonate and chloride, thereby detecting tendencies toward acidosis, chloride deficiency, and sodium depletion. Sodium being approximately the sum of chloride and bicarbonate in milliequivalents. Sodium chloride deficits are manifested clinically by early morning headache, muscular cramps, and dehydration. These may be abolished by administering 5 to 10 gm. of sodium chloride per liter of urine, which measure also stimulates thirst and promotes diuresis.

Until recently the importance of supplying liberal protein to patients with kidney disease was grossly overlooked. The protein stores of the body are depleted, as a rule, by long continued proteinuria. They must be replaced if osmotic equilibrium between the vascular and interstitial compartments is to be restored. Furthermore, a positive nitrogen balance is insurance against cachexia, lowering of antibody titers, and nutritional anemia. Nonprotein nitrogen should be carefully watched, but its elevation is not in itself a contraindication to a high protein intake, nor will the latter quantitatively affect the proteinuria.

There is no particular need to force protein in the absence of proteinuria or lowered plasma protein levels, and in view of Dock's⁵ experiments on rats it may be inadvisable. Dock states that although the extra oxygen consumed by the kidneys after changing from low to high protein intake is too small to be detected, the high protein diet leads to renal hypertrophy, presumably because of its content of aromatic amino acid. A very high protein intake may jeopardize recovery after some types of kidney injury. This statement is corroborated by Chanutin and Ferris.²³ The kidneys should not be spared at the expense of the rest of the body by a diet inadequate in protein. On the other hand, indiscriminate forcing of protein may delay the healing of renal lesions. An allowance of 1 gm. of protein per kilogram of body weight per day is ordinarily sufficient but not excessive.

The diet should be well stocked with vitamins and carbohydrate. Vitamin deficiency alone plays no demonstrable role in pathogenesis of renal disease, but it most certainly retards recovery and increases the patient's vulnerability to infection. Carbohydrate protects the liver, supplies readily available energy, and spares protein. Obesity is to be avoided, particularly if the patient is diabetic or hypertensive, but weight reduction should be gradual and effected at the expense of the fat depots.

Mobilization of Edema. The mechanisms involved in edema formation have already been discussed and considerable attention given to the regulation of water, sodium chloride, and protein in edema prevention and control. Edema and ascitic fluids are quite toxic and must be drawn back into the circulation slowly if untoward cardiac and central nervous system disturbances are to be avoided. Diuresis is promoted by increasing the renal blood flow, and thereby the number of functional nephrons, or by decreasing the reabsorption of water by the tubules. The mode of action and usage of various diuretics are discussed below.

Water is the safest, most effective, and least expensive diuretic available. Like alcohol, it presumably causes a temporary lowering of pituitary production of antidiuretic principle, thereby depressing tubular reabsorption. Diet-

talus has no diuretic effect in the absence of cardiac disease but in the aged nephritic with concomitant circulatory insufficiency digitalis may evoke profound diuresis. The *xanthine* group of drugs particularly caffeine is widely employed to increase urine volume. These compounds cause vasodilation of the afferent glomerular vessels and by increasing the number of functional glomeruli enlarge the filtering surface. *Sodium or magnesium sulfate* may be employed. The tubular epithelium is relatively impermeable to sulfate which is therefore readily excreted with large quantities of water. The sulfates however are not wholly safe in older patients. With delayed excretion sulfate accumulation can be dangerously toxic. Urea and ammonium salts which are converted into urea function upon the same principle. *Urea* one of the most efficacious diuretics also increases the glomerular filtration surface. The dosage is 20 to 60 gm daily but its administration is inadvisable if the serum non protein nitrogen is considerably elevated. In the presence of edema hypertonic glucose plasma acacia and serum albumin stimulate diuresis by drawing interstitial fluid back into the circulatory system and making it available for renal excretion. Acacia should be employed cautiously if at all because of its antigenic properties. *Protein hydrolysates* by increasing serum protein are more efficacious and much safer. Either plasma protein or hydrolysates of casein are now readily available. Various mercurial compounds act as diuretics by diminishing tubular reabsorption but their use is inadvisable in the presence of nephropathy.

Edema may be controlled then by regulating water and sodium chloride intake providing adequate dietary protein for plasma protein synthesis and administering certain diuretic substances. If these measures fail paracentesis thoracentesis or lumbar puncture may be necessary to relieve pressure upon vital organs. In elderly individuals particularly fluid must be removed slowly and in moderate amounts if shock is to be avoided. Intractable edema of the lower extremities frequently responds satisfactorily to incision and drainage with Southey's tubes the risk of infection being negligible even in debilitated patients.

Other Therapeutic Measures The management of *hypertension* and its consequences is fully discussed in Chapter 30. Concomitant nephropathy alters the patient's response to treatment however and materially influences one's selection of therapeutic measures. Drastic reduction of the arterial tension may precipitate renal decompensation and/or disturbing mental symptoms in elderly individuals. These difficulties subside when the blood pressure

persistent mild vasodilation but the therapeutic and toxic ranges are dangerously close and since the drug is normally eliminated in the urine the presence of nephropathy militates against its use.²⁷ When potassium thiocyanate is employed the blood plasma level should be carefully maintained at between 8 and 14 mg per 100 cc a dosage of 5 grains daily usually sufficing.²⁸ In the treatment of hypertension mild sedation is often desired.

family of drugs
glomerular filtra
tion rate of impaired

renal excretion, bromidism is a constant hazard if the dosage is not carefully controlled

Azotemia, as seen in geriatric medicine, almost always is caused by chronic, irreversible kidney damage. In a few instances it results from acute, reversible urinary suppression, incident to transfusion with incompatible blood, sulfonamide intoxication, hemolytic reactions, heavy metal poisoning, urologic surgery, and the like. These cases may be successfully treated with peritoneal lavage, utilizing the peritoneum as a dialyzing membrane until the kidneys are able to resume their normal excretory functions.³⁰ This is a formidable procedure but may be employed if other measures fail. Anuria accompanying acute diffuse glomerulonephritis is also sometimes amenable to surgical treatment. If it persists for four to six days in spite of medical measures, decapsulation of the kidney may prove to be a life saving operation. When azotemia is caused by chronic, irreversible renal disease, medical measures are indicated. Uremic symptoms respond as well to the correction of acidosis as they do to the reduction of plasma nonprotein nitrogen. In this stage of the disease, phosphates and sulfates are elevated while blood chlorides are lowered, and the alkali reserves are depleted. An alkaline ash diet is indicated, and additional alkali may be given orally or parenterally. In emergency situations, 200 cc. of 4 per cent sodium bicarbonate may be administered every twelve hours until proper acid base balance has been established.³¹ The correction of acidosis may prevent or relieve the intractable vomiting so characteristic of azotemia, and although not a curative measure, frequently enables the patient to spend his last days in relative comfort. Uremic convulsions are relieved by lumbar puncture, the intravenous administration of hypertonic solutions, and sedation. Intravenous magnesium sulfate must be used with caution, however, in geriatric patients, because impaired renal excretion of the magnesium ion causes its rapid accumulation and may provoke profound depression. The therapy of azotemia resulting from chronic kidney disease is, then, symptomatic at the present stage of medical development. About all one can do is make the patient as comfortable as possible during his last few days.

Renal decompensation like cardiac decompensation, is not necessarily fatal. Compensation may be restored even in longstanding and extensive renal disease, but the functional reserve never goes back to its previous level, the margin of safety gets smaller and smaller until there is none. The greatest number of attacks of "uremia" or renal decompensation that we have observed in one patient is four, survival with lowered reserve followed the first three episodes, the last was fatal. In contrast we have seen patients survive as many as nine episodes of acute congestive cardiac decompensation to die in the tenth attack. Age *per se* reduces the functional reserve and thus renal damage in later years always responds poorly to treatment. The essential failure, in most instances, is the inability of the kidneys to concentrate the urine. An ever increasing urinary volume is necessary to get rid of metabolic debris. Ultimately the limit of circulatory endurance is reached and final failure is as often as not due to cardiac exhaustion.

SUMMARY

The fine line of demarcation between normal and pathologic renal change with advancing age cannot be defined. Nor are many of the etiologic factors

in nephropathy clearly understood. Further research is urgently needed in this important field, for the causes of disease must be known if therapy is to be intelligently planned and effectively executed. Renal disease in the senescent and senile must be discovered before it becomes obvious if irreparable renal damage is to be minimized. Thorough physical examinations and urinalyses on periodic health inventories should be coupled with renal function tests. Once the diagnosis has been made, treatment must be directed toward correcting concomitant disorders as well as ameliorating renal dysfunction. Every effort should be made to restore physiologic equilibria, which are so readily disturbed in aged individuals, and to prevent further kidney damage. It is difficult to restore renal functional capacity if the accumulative damage is too extensive. The effects of renal inadequacy involve all the structures, tissues, and functions of the patient. Treatment of renal disease, perhaps more than any other therapeutic problem, involves treating the patient rather than the disease.

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CHAPTER 38

SURGICAL RENAL DISEASE

ROBERT H. HERBST AND JAMES W. MERRICKS

THE operative correction of kidney and ureteral conditions may be as successful in elderly people as in younger patients. As the age of the patient rises so does the probability of renal, cardiovascular and pulmonary disease. However, it must be recalled that it is not the age of the patient, but the degree of degenerative change present, that should influence judgment concerning the extent of surgical intervention necessary in the management of this type of patient. Only rarely does the elderly patient require surgery on the kidney as an emergency measure. It is possible to study the upper urinary tract with great accuracy and deliberation at all age levels.

The kidney is the only organ in the whole genitourinary tract necessary to life. This fact puts added emphasis upon associated study of the cardiac status, the relative importance of the blood pressure level and condition of the blood vessels, pulmonary function, foci of infection, and degree of possible anemia. It is difficult to draw a dividing line beyond which the urologic patient should not be subjected to upper urinary tract surgery. In general, if life expectancy seems reasonably good, if the patient has been leading an active life, and if urologic surgery offers the only chance of relief, one should not withhold operative interference.

TUMORS OF THE KIDNEY

The most important surgical disease of the adult kidney is tumor. Kidney tumors comprise approximately 0.5 per cent of all tumors. Almost all new growths of the kidney are malignant. Therefore, when signs point toward a renal tumor, cancer is the first thought in the older patient. Early diagnosis and nephrectomy, if metastases have not occurred, offer the only chance for cure. Death from primary renal neoplasm may occur without characteristic evidence of renal involvement. The patient may present metastatic lesions in the lung, liver, or brain, the original site being found only at necropsy. Five-year control of renal tumors treated by nephrectomy has been improved from 15 per cent¹ to 38 per cent², principally because of earlier and better diagnosis. In too many cases the diagnosis is delayed by failure to make a proper urologic examination.

About 94 per cent of renal tumors cause hematuria as the earliest and most prominent symptom. Even so, bleeding occurs relatively late in the course of the disease. Therefore, the source of any urinary bleeding should be investigated immediately.

The other symptoms pointing directly to the kidney are mass in the flank and pain. Pain is not common early. It may be colicky, due to clots passing down the ureter, or more or less steady, due to the tumor itself. Indirect

in nephropathy clearly understood. Further research is urgently needed in this important field, for the causes of disease must be known if therapy is to be intelligently planned and effectively executed. Renal disease in the senescent and senile must be discovered before it becomes obvious if irreparable renal damage is to be minimized. Thorough physical examinations and urinalyses on periodic health inventories should be coupled with renal function tests. Once the diagnosis has been made, treatment must be directed toward correcting concomitant disorders as well as ameliorating renal dysfunction. Every effort should be made to restore physiologic equilibria, which are so readily disturbed in aged individuals, and to prevent further kidney damage. It is difficult to restore renal functional capacity if the accumulative damage is too extensive. The effects of renal inadequacy involve all the structures, tissues, and functions of the patient. Treatment of renal disease, perhaps more than any other therapeutic problem, involves treating the patient rather than the disease.

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Fig. 124 Multiple intrarenal cysts of left kidney in woman, seventy four. Excretory urogram. Widespread distortion of pelvis and calyces and hematoma.



Fig. 125 Same patient as Figure 124. Retrograde pyelogram suggests less distortion. At operation the kidney was large and felt quite hard.

The pyelographic picture shows a filling defect in the pelvis with minimal or no distortion of the calyces, presenting quite a different picture from that of

parenchymal tumor These pelvic tumors resemble ureteral and bladder tumors in general

Wilms's tumor, the common renal tumor of childhood, is extremely rare in older patients This may be a rapidly growing tumor, attaining considerable size seldom producing hematuria The general color of the tumor is gray as opposed to the yellowish tint of the hypernephroma It consists of various types of embryonal tissues

Treatment The only known control of kidney tumors is surgical removal The problem of each patient must be considered individually Nephrectomy is the treatment of choice in the absence of demonstrable metastases However should tumor arise in a patient with but a single kidney, treatment necessarily must be conservative We have just such an instance under observation



Fig 126 Papillary tumor of renal pelvis retrograde pyelogram Filling defect in pelvis with little distortion of calyces Hematuria Nephroureterectomy was performed and the patient is living twelve years later There was one recurrence in the bladder nine years ago

at the present time The right kidney mass was discovered at laparotomy for gallbladder disease No left kidney was palpable and none found on complete urologic study Exploration of the right single kidney showed extensive involvement with tumor X ray therapy has reduced the size of the tumor and not affected the renal function over a period of a year, an unusual result

It is often as difficult to offer an accurate prognosis even after nephrectomy The immediate surgical mortality should be very low In the patient of advanced age the parenchymal tumor, if not removed, may grow very slowly while the patient succumbs to other disease processes Death from unoperated tumor, however may occur in a few short months after the first symptoms appear In general, renal tumor follows the pattern of decreased virulence or malignancy in the aged compared with younger individuals Metastases in

distant organs, commonly lung and bone, are usually contraindications for nephrectomy. Long survival times have been reported, however, following removal of the kidney tumor along with a single metastasis in a lung. It may be necessary to remove the kidney for severe hemorrhage even in the presence of known metastases. Other factors affecting survival include invasion of the renal vein. Fully half of the patients should survive five years if the renal vein has not been involved. Actually this figure is about 38 per cent, probably because of some lymphatic spread of tumor, in addition to involvement of the vein.

The outlook for papillary tumors of the renal pelvis is definitely better if, in addition to nephrectomy, complete ureterectomy is done. Papillary pelvic tumors tend to extend down the ureter. Follow-up cystoscopic examinations should be carried out at intervals for some years after nephroureterectomy to detect bladder implantation recurrences as early as possible. The prognosis for squamous (infiltrating) carcinoma of the renal pelvis is very poor. This rapidly invading tumor is frequently associated with stone and chronic infection. The foregoing renal tumors are very rarely bilateral.

Benign Tumors. Benign tumors of the kidney occur chiefly as small multiple adenomata bilaterally and most often are found coincidentally at necropsy. Only rarely does an adenoma grow to sufficient size to warrant surgical exploration. Also very rare are fibromata, lipomata, fibrolipomata, and cystadenomata in the adult kidney. Fat replacement of the kidney, usually associated with stone and infection, is unusual, but may simulate tumor in the pyelogram. The prognosis in these conditions is excellent except for the slightly more malignant cystadenomata.

CYSTS OF THE KIDNEY

Cystic disease of the kidney in the aged is of importance because of the occasional difficulty in clinically differentiating solitary, multiple, or multilocular cysts from tumors, and because the size of the kidney mass, with or without pain, may justify surgical excision of the cyst or nephrectomy. Cysts in the midportion of the kidney are most likely to destroy the renal paren-

at nephrectomy. Cysts of the pancreas and liver, although rare, may simulate cysts of the kidney (Figs 127, 128) in x-ray films.

Polycystic disease of the kidneys is mentioned merely to lend completeness to the discussion. The average patient with this disease seldom attains advanced age. We have under observation a family of six, the two oldest of whom, the mother and aunt, lived sixty-six years with polycystic kidneys, finally succumbing to renal failure. Most of these kidneys are bilaterally palpable, but should seldom offer diagnostic difficulties except in differentiating

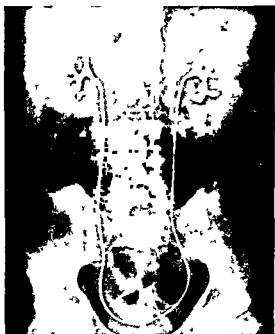


Fig 127 Solitary cyst of lower pole of left kidney causing only slight spreading of lower calyces in patient sixty-one years of age. Resection of cyst with kidney preserved.



Fig 128

Fig 128 Cyst of liver simulating cyst of lower pole of right kidney



Fig 129

Fig 129 Showing hand pushing cyst shadow away from intact lower pole of right kidney. Same patient as Figure 128.

later, unoperated, she passes an occasional daughter cyst in the urine. X ray therapy has not been considered advisable for destruction of the ova because of the probability of reaction.



Fig 130 Polycystic disease of kidneys retrograde pyelograms Calyces of left kidney show more distortion and elongation than those of right



Fig 131



Fig 132

Fig 131 Huge hydatid cyst of left kidney retrograde pyelogram normal right pyelogram Treatment was pelvic lavage with sodium sulfathiazole solution intermittently for one year

Fig 132 Retrograde pyelogram four years later in same patient as in Figure 131 Note calcification in cyst Daughter cysts now rarely passed in urine six years later

RENAL AND URETERAL LITHIASIS

Some older patients live out their lives comfortably with stones in one or both kidneys. They may experience an occasional attack of pain and fever without serious upset. Not infrequently such a patient will be living with one kidney containing a stone, the opposite kidney having been removed previously for stone.

Surgical manipulations of a single kidney, especially nephrotomy for extensive lithiasis, are poorly tolerated by elderly patients. Nephrectomy is at times preferable to extensive exploration of the kidney in search for multiple calculi, provided the opposite kidney has sufficient functional reserve. This attitude is almost a complete about-face from the accepted management of stone in young patients. Pyelotomy for stone is the most common conservative operation upon the kidney in older patients.

The tendency for these solitary calculi to recur is decreased in the elderly. The majority of stones seem to form during the more active years of life, hence most of those seen in the elderly have been present over a long period of time, either because they were asymptomatic, or because removal was postponed through the wishes of the patient or because of prior poor general physical condition. We have followed a small group of such patients for years, watching their stones remain about the same. It is desirable to repeat x-ray examination in all patients with stone every few months. After repeated recurrence of renal calculus colic, cooperation is usually easier to obtain. In the cooperative patient, conservative management affords excellent opportunity for attempts to prevent further growth of stones or to dissolve them by conservative nonsurgical methods. Encouraging reports of reduction of the size of stones or their actual disappearance through dietary means and irrigations with various acids appear in the literature from time to time. Up to the present we have been unable to accomplish significant reduction in size of renal calculi by such means, although we have been fairly energetic in our attempts. It is said one must almost live with such patients, supervising their every move.

The advent of newer *drugs* and *antibiotics* for controlling infection will reduce the incidence of recurrence of calculi after surgical removal, especially when urea splitting organisms such as *Proteus vulgaris* are present. Streptomycin has been particularly valuable in such infections. However, sufficient dosage to control the infection may produce toxic effects. Delayed elimination of foreign substances must be expected in the elderly. Modern control of infection has definitely enhanced the safety of more conservative renal surgery. Kidneys may be saved today that would have been removed formerly.

Metabolic errors may cause stones such as cystine and urate calculi without gross structural abnormality. Recently a middle-aged patient came to necropsy after years of bilateral postoperative recurrences of renal phosphate calculi. No urinary obstruction existed, there was little infection, and no parathyroid disorder could be found, yet at no time during our years of clinical observation could this patient's urine be acidified. Hence the assumption may be made that an error in ammonia metabolism existed in the kidney.

Stones in the kidney, secondary to a primary endocrine disease occur in the

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removal of a parathyroid adenoma reduced the blood calcium level to 6.5 mg per 100 cc. Tetany did not occur. The obstructing prostate was removed by endoscopic resection. Some months later, after returning home, the patient succumbed to renal failure.

Stone in the ureter is neither so common nor so important in elderly as in younger or middle-aged patients. In older patients, ureteral stones may grow to large size but because of their very slow growth become rounded and smooth. It is amazing how such large stones can and do pass spontaneously down the ureter. Nevertheless, manipulation of stone may result in ureteral perforation, more frequent in the old than in the young.

Spontaneous perforation of the renal pelvis or ureter is observed occasionally in older patients. An interesting group of such perforations is reported by Herman *et al*.³ The oldest patient, a woman eighty, presented a preformed periureteral abscess at the site of an impacted stone. At cystoscopy a ureteral catheter was deflected by the stone into this abscess. Recovery followed incision and drainage of the abscess with no urinary leakage. Acquired ureteral diverticulum is usually associated with impacted ureteral calculi.⁴

Instrumental manipulation for loosening or extraction of lower ureteral stones always requires fine judgment and skill. Injury from such procedures is more common in the patient of advanced age. Much safer is surgical removal of impacted ureteral stones which cause symptoms, renal injury by obstruction, or both.

LOCAL DISEASES OF THE URETER

The ureter is rapidly becoming an object of major interest to the urologist. It must be dealt with somehow before the bladder is resected partially or *in toto* for carcinoma. One ureter may be tied off deliberately while the other is transplanted to skin or bowel preparatory to cystectomy. One ureter may be tied off deliberately during resection of the large bowel. Only occasionally does the resulting obstructed kidney require removal subsequently, if originally uninfected. Ureteral injury during the course of gynecologic surgical operations is the most frequent of all surgical injuries. It must be remembered that the vast majority of these mishaps occur in young or middle-aged women. Ureteral avulsion during renal operations is very rare. Ureteral injury due to

aged. We have followed such a patient for seven years, at the age of eighty, reimplantation of the ureter was carried out successfully preliminary to a suprapubic enucleation of a 240 gm prostate.

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tion

ureter. Metastatic tumors of the ureter are quite rare, originating chiefly from kidney, bladder, prostate, stomach, and uterus.

RENAL MOBILITY

The kidney which is displaced because it is abnormally movable is easily distinguished from the displaced, fixed (ectopic) kidney because the former

can be moved back into the normal renal bed. The kidney is not a fixed organ, but normally moves with respiration and change in body position. Perirenal fat and fibrous tissue layers serve to keep the kidney in position. These supports often become lax with advancing years and/or extensive weight loss. Nephroptosis may occur at any age, but increases in frequency toward middle life. Ptosis of other abdominal organs may accompany the dropped kidney, but is not constant. Symptoms and findings vary widely. Pain may be out of proportion to the degree of ptosis or to the demonstrable damage suffered by the kidney. *If the ureteropelvic relationship is not disturbed by the change in position of the kidney, usually little damage due to renal retention can be demonstrated.* Pull on the peritoneum or bowel by the kidney may cause gastrointestinal symptoms. The patient may be nervous and ill because of



Fig 133



Fig 134

Fig 133 Infected right hydronephrosis and ptosis of both kidneys in woman seventy three years of age. Plastic repair of right kidney and nephropexy.

Fig 134 Same patient as Figure 133. Excretory urogram one year later shows kidney in good position with excellent function and good result.

discomfort due to traction or as the result of infection which so often supervenes in the poorly draining kidney.

Palliative treatment deserves consideration in the majority of these patients. A well fitted two way stretch girdle with a small supporting pad centered just medial to the anterior superior iliac crest provides excellent support for the kidney. The girdle must be put on with the patient recumbent. It is pulled up over the hips which are supported on pillows as the breath is exhaled. Then a pad either pneumatic or fashioned from one or two ordinary covered cotton dress shoulder pads is slipped into place under the girdle

normal placement of the ureter, and the like) must be considered when con-

servative treatment affords no relief Where properly indicated, nephropexy is a very rewarding operation Care must be taken not to try to fix the kidney too high, especially on the right side, because the weight of the liver may break the fixation down

THE OBSTRUCTED KIDNEY

A definition of terms is in order here By far the most common cause of urinary retention in the kidney is bladder neck obstruction, benign or malignant, less frequent are *extraurinary tumors in the renal pelvis and bladder tumors* However, we are not concerned here with these particular obstructive lesions, but with obstruction high up in the urinary tract

In addition to abnormal renal mobility with ptosis, primary noncalculous hydronephrosis of local origin may be caused by abnormal blood vessels to the lower pole of the kidney pressing upon or distorting the upper ureter, abnormal placement of the ureter into the renal pelvis, periureteral bands, and ureteral kinks or stricture Infection is not uncommonly present in the long standing hydronephrotic kidney Occasionally one encounters a large hydronephrosis in an old patient who comes to the doctor for mild pain in the side or back with no history of infection and with urine clear of pus The management of hydronephrosis is influenced by the condition of the patient in relation to his life expectancy If symptoms indicate the necessity of surgery, nephrectomy is the operation of choice Numerous instances are on record of long survival after removal of giant hydronephrotic kidneys

Conservative operative procedures, such as freeing of bands, severance of small abnormal vessels, and nephropexy, may be undertaken even if the opposite kidney is adequate, but long involved plastic procedures are hardly indicated for those of advanced age If the function of both kidneys is reduced, nephrostomy under local anesthesia may be a life-saving procedure If the patient improves and conditions warrant, a secondary repair of the kidney may be attempted, but is seldom justified

Drainage of an obstructed, infected kidney may be done by indwelling ureteral catheters over long periods of time We have kept ureteral catheters, with occasional changes, in the kidney of a sixty seven year old man for almost four months until his renal function improved enough to allow removal of a calculus causing hydronephrosis in his single kidney

In general, nephrectomy at the advanced age level is the procedure of choice for the symptom producing hydronephrotic kidney and is distinctly necessary when the kidney is badly infected, if the function of the opposite kidney appears to be adequate With the advent of chemotherapy and the antibiotics, control of infection in the kidney is now easier This one factor has been responsible for much greater success with surgical correction of hydronephrosis, for infection has always been the stumbling block to good results in such plastic surgery (Figs 133 and 134)

RENAL INFECTIONS

In the aged, as in the young, renal infections are probably not so common today as they were years ago because of the widespread use of the newer antibiotic drugs, which are useful in infections of the genitourinary tract as well as elsewhere This applies particularly to those infections unaccompanied by stone or obstructions, such as prostatic enlargement, which is a prominent

cause of upper urinary tract infection in the male. In the female cicatricial urethritis may lead to bladder and kidney infections by disturbing the proper drainage of those organs. Fulminating infections still occur when the causative organisms do not respond to any urinary antiseptics. Hematogenous spread of infection may result first in focal nephritis then pyelonephritis with or without formation of cortical abscesses and finally perinephric abscess. Such a chain of events can occur rapidly in the elderly and the picture may be most confusing and obscure, the true nature of the disease being revealed only at necropsy. Recent experience indicates that cortical renal abscess and carbuncle which are not common in the elderly respond to currently available urinary antiseptics and antibiotics. Early diagnosis of perinephric abscess is



Fig. 135 Tuberculous calcified pyonephrosis with functionless left kidney excretory urogram. Normal right kidney. Frequency of urination a recent symptom. Nephrectomy was carried out with recovery.

still difficult because the onset is so insidious and indefinite. Localizing signs become obvious only in the later stages of the disease when extension of the abscess and overwhelming toxemia are prone to occur.

If renal infection does not continue to the formation of abscess but persists as a low grade inflammatory reaction with or without occasional exacer-

Pyonephrosis, the late stage of chronic renal suppuration, usually arising from long continued urinary tract obstruction, requires nephrectomy for relief. Judgment is necessary in deciding whether the patient can survive such a difficult procedure. In the elderly, pyonephrosis may occasionally be due to tuberculosis. At times no other active clinical focus can be found in the body, but it must be remembered that active pulmonary tuberculosis in older men and women is not uncommon (See Chapter 22). Autonephrectomy is the term applied to the process which follows a walled off chronic infection with the ureter remaining securely sealed. The usual course of events is not quite so fortunate as in autonephrectomy. A little leakage of infected drainage from the tuberculous kidney may occur down the ureter, causing at first a mild, and then persistent, painful cystitis with distressing frequency of urination. It is this frequency which finally brings the patient to the doctor. Discovery of the tubercle bacillus in the urine may be very difficult. Removal of these infected, sac like kidneys may be done easily or may prove fatal, depending upon the degree of attachment to adjacent organs and the nutritional and cardiovascular reserves of the patient. Suppuration in and about the kidney may progress to a remarkable degree in the aged without the usual obvious findings of pain, fever, and leukocytosis (Fig 135).

VASCULAR LESIONS OF THE KIDNEY

Rarely is a vascular disturbance of the kidney of surgical significance. Thrombosis of the renal vein and thrombosis and embolism of the renal artery are largely medical problems. Urologic study serves, where necessary, to distinguish these diseases from primary renal disease. The symptoms of pain

in the loin, and fever, and leukocytosis, are not diagnostic of these conditions.

Aneurysm of the aorta, either the massive or the mycotic dissecting type may cause pain simulating renal colic (See Chapter 28). We have recently observed patients presenting just such diagnostic problems. One a man of sixty seven had severe right sided pain and fever. Complete urologic study was negative, yet a general surgeon explored the perirenal region with both needle and scalpel, encountering a diffuse aortic aneurysm which subsequently caused the patient's death as proved by necropsy. Another man, seventy two, had left loin pain and fever. Urologic findings were negative. Death occurred shortly with no more definitive signs. Necropsy revealed rupture of the abdominal aorta due to dissecting aneurysm, with massive perirenal hematoma.

Perirenal hematoma, a rather unusual condition, results from rupture of an aortic or, more rarely, renal artery aneurysm and other conditions, such as extravasation of urine from urinary stasis due to bladder neck obstruction, stone, hydronephrosis, infection, or trauma causing hemorrhage. Sudden pain, shock, and a rapidly increasing mass in the loin should point to the diagnosis. Immediate exploration is indicated.

Aneurysm of the renal artery may be suspected because of a shadow of calcification in the aneurysm wall on a roentgenogram of the kidneys (Figs 136 and 137). Symptoms are rare when calcification occurs. Rupture leads to acute pain in the flank and evidence of perirenal hematoma.



Fig 136



Fig 137

Fig 136 Calcification of aneurysm of right renal artery (symptomless)

Fig 137 Same case as Figure 136 Excretory urogram showing relation of aneurysm to normal renal pelvis

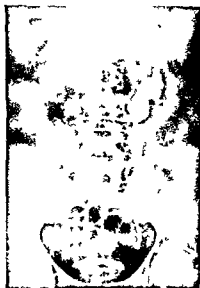


Fig 138



Fig 139

Fig 138 Horseshoe kidney with stone in left renal pelvis, in patient aged sixty four years excretory urogram

Fig 139 Same case as Figure 138 Retrograde pyelogram showing lateral position of ureter Pyelotomy was carried out with removal of calculus and division of isthmus Many abnormal vessels were present

ANOMALIES OF THE UPPER URINARY TRACT

Abnormalities of renal development most often become evident early in life and therefore rarely present diagnostic difficulties in later years. The most common anomaly, duplication of the renal pelvis with incomplete or complete double ureter, frequently leads to infection and stone formation in the smaller upper segment, with recurrent attacks throughout the patient's life. Incontinence results if one ureter opens extravesically in the female. Occasionally heminephrectomy, ureteroureterostomy, or nephrectomy may be required later.

Occasionally an ectopic kidney is discovered accidentally late in life. We have encountered a patient who had borne five children without difficulty. Her ectopic right kidney rested in large part over the bony pelvic brim near the posterior midline.

Unilateral fused kidney presents a flank mass occasionally suggestive of renal tumor but should be easily distinguished by pyelographic study. Congenital absence of one kidney has proved disastrous when nephrectomy has been carried out on the only existing kidney. Nephrectomy should be invariably preceded by demonstration of functional activity of the other kidney. Failure to demonstrate effective urinary secretion on the other side is an absolute contraindication to nephrectomy. Horseshoe kidney with stone in one or both pelves presents a difficult problem in insuring adequate drainage post-operatively to lessen infection and prevent recurrent stone (Figs 138 and 139). Variations in renal rotation and blood vessels are common and may cause considerable difficulty in exposing and carrying out various operative procedures on the kidney.

INJURIES OF THE KIDNEY

The kidney is well protected and cushioned against injury by being situated deep beside the spine, under the ribs, and is usually surrounded by fat and covered with the heavy muscles of the back. Violence severe enough to lacerate the kidney perilously must be direct and usually results in the fracture of overlying ribs. In general most ruptures of the kidney occur in industrial accidents in young, active patients. In the elderly, falls in the home and traffic mishaps account for most injuries of this type.

The most prominent symptom of renal trauma is hematuria. Perirenal hematoma and shock are added to the picture in deep lacerations of the kidney. Associated visceral and skeletal injuries must be evaluated, bearing in mind that the older patient may not tolerate extensive injuries as well as younger and more robust subjects. The outcome of such accidents depends in great measure upon the health of the patient prior to the trauma.

In our limited experience, surgical interference is seldom necessary for renal trauma unless complicated by continuing hemorrhage. Needless to say, constant attention is necessary, not only to follow the course of the renal bleeding but to treat the other injuries. Blood transfusions are of paramount importance. (See Chapter 8.) Excretory urography is chiefly of value in revealing an intact kidney. It is not possible to delineate varying degrees of severe renal rupture on the excretion urogram. Cystoscopy and retrograde pyelograms have no place in the management of the acute phase of renal trauma, but should be reserved as an aid in the imperative follow-up study each patient should have later to determine the residuals of injury, namely

stone, hydronephrosis, cystic change, and infection. Subsequent surgical procedures, including nephrectomy, may be necessary. Accurate urologic study throughout the course of treatment of trauma to the urinary tract is just as essential as repeated roentgenologic study of the fracture patient.

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CHAPTER 39

DISEASES OF THE URETERS, BLADDER AND URETHRA

FRANK HINMAN AND FRANK HINMAN, JR

To provide orientation for this discussion the urologic diagnoses of all patients sixty years and over seen in the clinic and hospital of the University of California have been reviewed 2900 in all Of these, 927, or about a third, concerned the urethra and penis (193), the bladder (654), and the ureters (80) We shall try to devote space to each disease in proportion to the frequency with which it was encountered rather than to attempt a sketchy survey of all diseases of these organs, which information can be found in an urologic text book We have tried to answer the questions What are the urologic diseases of the aged? What factors predispose to them? What is the differential diagnosis in older patients and, in what way does the treatment differ from that of younger individuals?

THE PENIS

In our series the principal diseases of this organ were inflammatory lesions of the prepuce and carcinoma

Preputial Inflammation Few men reach sixty years unable to retract the prepuce, yet older men may have acquired *phimosis* and, because of their di-

the there is a tendency to the development of a chronic inflammation

men of good hygiene with cleansing and drying of the glans and prepuce before replacing will often relieve the inflammation and prevent recurrence Should the prepuce not be retractile it is necessary to perform a circumcision

croid Although circumcision is inadvisable in the presence of inflammation, it may be indicated later Frequently the dorsal slit accomplishes as much as a circumcision

P

cular changes of age added to the fact that the prepuce is often of a thickened and inflamed condition

sal longitudinal incision of the tight section of the prepuce will allow reduction Attention must be paid to the immediate reduction of the foreskin after examination or catheterization in elderly men for paraphimosis readily follows

Penile Tumors. Twenty per cent of the penile lesions diagnosed in this series were carcinoma, but they formed less than 0.1 per cent of all urologic diagnoses. The sixth decade has the highest incidence but this tumor is not uncommon in later years.

Etiologically, chronic irritation secondary to phimosis is the most potent predisposing factor. Members of religious groups circumcised at birth rarely are found with penile cancer, but circumcision after infancy does not necessarily prevent the disease. It forms from 1 to 3 per cent of all cancers in the European male. A few cases arise in scars and venereal ulcers, and after phimosis with balanitis.

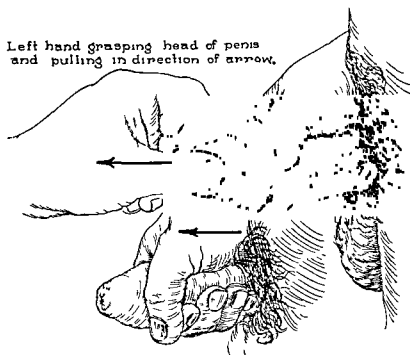


Fig. 140 Method of reposition of paraphimosis. The glans is seized with thumb and forefinger of the left hand and the penis drawn out. The edema and swelling of the prepuce are massaged by gripping the shaft with the right. On releasing the left hand the glans usually will slip beneath the prepuce. (After Steinmann.)

Pathologically, the lesion like other skin cancer begins as a wart or eroded papule, as a keratosis, or as a definite ulcer. The patient usually describes a small "sore" which fails to heal or a persistent discharge from beneath the prepuce. Even though 69 per cent of lesions are on the glans, they exist on an average of two years before diagnosis.

The *diagnosis* may be difficult because of the similarity between benign and malignant warts, and although the benign lesions are softer and less friable, biopsy must be done before surgery (Fig. 141). It is necessary to watch

lesions of the penis Its presence in connection with carcinoma is indicative of metastasis in at least one-third of the cases

The principle of treatment is adequate early excision In those with a small early lesion, however, encouraging results with radium have been achieved by Dean In lesions larger than 2 cm, radiation therapy is of little value even as palliation and often results in a painful debilitating mass Cure by surgery is relatively frequent In the average and operable case, the penis is amputated by a margin of at least 2 cm Total emasculation is rarely indicated The question of the value of removal of regional nodes by radical groin dissection is difficult to answer The lymphatic drainage from the penile tissues superficial to Buck's fascia is to the inguinal and superficial femoral nodes, but with involvement of the corpora the drainage is the same as that of the urethra and hence is in large part to the deep iliac nodes The criteria

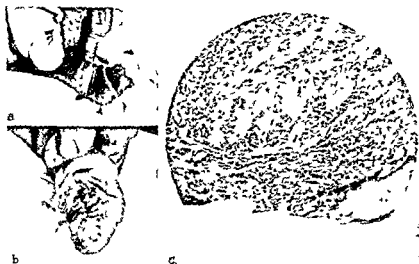


Fig 141 a Gross appearance of a carcinoma of the penis b With prepuce retracted c Microscopic picture (Hinman Principles and Practice of Urology)

for choice of patients for radical groin dissection are far from agreed upon Probably if no nodes or nodes less than 1 cm in diameter are present, the operative mortality exceeds the possible benefit except in the better surgical risks, these patients should be observed at monthly intervals for a year and bimonthly thereafter for palpable regional involvement (Taylor and Nathanson) When larger lymph nodes are palpable, the probability of metastasis is high and a bilateral block dissection of the inguinal, femoral and, if possible external iliac nodes is indicated It is often best to use two stages, amputating the penis in the first so that the field will be clean for the second

Without treatment, the course of the disease is slow, with most patients living from one to three years before reaching a critical condition (Barney)

THE MALE URETHRA

Urethral Stricture was the most common disease of the male urethra, and formed 20.8 per cent of urethral disease even when cases of both sexes were

included. About a quarter of the strictures in our series were traumatic or postoperative while the remainder were inflammatory. With the present antibiotic therapy of specific urethritis, postgonorrheal strictures will become limited more and more to the older age group, who received inadequate or traumatic initial treatment. Stricture has become increasingly frequent following the use of the retention catheter or following transurethral resection. The febrile response with the former, the secretions at the meatus and the severe upper tract infection which follows, make this a source to be feared. The use of a smaller catheter will be a help in prevention, but occasionally only performance of a suprapubic cystostomy will allow the infection to subside. The increasingly common postresection strictures are due to forced dilation before insertion of the resectoscope. Unless the instrument slips easily through the penile urethra, perineal urethrotomy is indicated (Nesbit). All patients should have sounds passed two or three weeks after resection to detect strictures. A prolonged course of dilation may be necessary.

The *pathologic changes* in the urethra are most commonly located in the bulbous portion and are usually single annular constrictions if of traumatic origin and multiple if inflammatory or chemical.

Symptoms may be absent and the stricture only found after marked renal damage has occurred, although chronic urethral discharge and decrease in size and force of the stream, with dribbling, frequency, and retention of urine are the usual complaints.

The *complications* of urethral stricture are serious. Like the obstructing prostate, a stricture may cause back pressure with hydronephrosis or may maintain infection in the middle and upper tract with resulting chronic and progressive pyelonephritis. These are only partially reversible on treatment of the stricture. The obstruction and infection also causes dilation and diverticula of the urethra, with occasional rupture and resultant serious periurethral phlegmon. Infection of the prostate, seminal vesicles, epididymis, and other local structures are often found back of a stricture. Hemorrhage, either spontaneous or commonly after instrumentation, may be severe and necessitate suprapubic cystostomy if not controlled by retention catheter and local pressure.

The *diagnostic measures* should be preceded by preparation unless the patient is in retention. Irrigations, massage, diathermy, sitz baths, and urinary antiseptics should come before any instrumentation. Exploration is then carried out with well lubricated bulbous bougies, working from 6 to 26 F. Additional information may be gained from a urethrogram. Anterior urethral obstructions in elderly men without antecedent history are suggestive of carcinoma, the carcinoma itself forming the stricture. Also over one fourth of carcinoma of the male urethra arises in old strictures.

Treatment is a continuation of the diagnostic instrumentation. If the stricture is within 2 cm. of the meatus, meatotomy and incision are indicated. If in the pendulous urethra or bulbous urethra, gradual dilation at intervals of three to seven days is usually the treatment of choice. If a well lubricated,

stricture upon healing. It is a good rule to increase the size of the instrument

only two sizes on the French scale at each dilation. When the stricture admits the largest sound that will pass through the uninvolved portion, the interval between dilations should be doubled until the time of recontracture is determined. With full urethral dilatation, this period is generally between four and twelve months.

Recontracture of strictures is the rule, but control by dilatation is generally preferable to operative intervention. Operation rarely cures a stricture unless a resection and end-to-end anastomosis can be performed. Internal urethrotomy consists of blindly cutting a stricture in the dorsal portion of its circumference with a knife designed for the purpose passed through the urethra. This operation does not cure the stricture but makes it more readily dilatable. There is a fairly high incidence of postoperative bleeding and sepsis. Its principal value is in the treatment of tight strictures of the pendulous urethra.

Acute retention caused by stricture is the result of sudden inflammatory occlusion of the urethra. Voiding is rapidly resumed when the edema lessens, even though a filiform sized stricture is still present. The retention may be relieved by a hot sitz bath and an opiate. If these fail, the passage of filiforms should be attempted. A small hollow follower is then passed to empty the bladder and the filiform tied in place in the urethra. If the follower will not pass, voiding around the filiform will almost always occur. Usually the edema will subside sufficiently in twenty four hours so that the inlying filiform is unnecessary. The use of steel sounds or catheters is productive of much urethral trauma and rarely succeeds in relieving retention. The same may be said about attempting to pass instruments under general or regional anesthesia. Should the stricture be impermeable to filiforms, cystostomy is indicated.

Urethritis in the male is relatively uncommon in the aged, and the incidence of gonorrheal and syphilitic urethritis is greatly decreased. Nonspecific urethritis is the common type, especially in debilitated men, and is usually due to temporary pathogenic activity of the pyogenic organisms always present in the anterior urethra. Sudden change in sexual habits as on the death of the wife may likewise lead to nonspecific, often abacterial, inflammation of the urethra, and of the prostate and vesicles as well.

The *diagnosis* is made by the presence of purulent urethral discharge which contains mixed organisms or no organisms on smear and which on repeated examination is shown to contain no gonococci or tubercle bacilli. Tumor, syphilis, stone, and chemicals must be ruled out as causes and the urethra should be examined for stricture. In approximately 75 per cent of the cases, urethritis is secondary to nonspecific prostatic seminal vesiculitis (Pelouze). Following treatment of these conditions, the urethritis will subside.

The *treatment* must be mild, for the mucous membranes of these elderly patients are sensitive to chemicals and trauma. Irrigation with 1:8000 per manganate solution, perhaps alternating with 5 per cent mild silver proteinate solution, should clear the symptoms in five or ten days. If a favorable response is not had after three or four weeks of such treatment, endoscopy should be done for further study, the reason usually lies in some overlooked factor in the bladder, prostatic urethra, or bulbomembranous urethra.

Neoplasms of the urethra formed an unexpectedly large percentage of all urethral disease (6 per cent) in this age group but a smaller proportion were in the male.

Squamous cell carcinoma was found in over 90 per cent, as is consistent with published series. In the male it arises most commonly in the bulbourethral glands, less often from the glands of Littre, and is distributed rather uniformly throughout the length of the urethra with the exception of the fossa navicularis where it rarely occurs. These are somewhat more malignant than penile tumors, and tend to metastasize to the pelvic nodes rather than to the inguinal region.

The *symptoms* are those of urethral stricture with obstruction and infection. Often by the time that the patient is seen a definite mass may be palpated, and all too often there is ulceration, fistula, and hemorrhage. In the late stages, infection leads to death. Biopsy by endoscopy must be done if the diagnosis is in doubt.

The *treatment* is excision with a good margin even if cystostomy and extensive urethrectomy are required. In some cases, transurethral resection of the infiltrating portion with radiotherapy is indicated. The prognosis is very bad.

THE FEMALE URETHRA

Diseases of the female urethra differ from those of the male urethra as much as the anatomy differs. Stricture is very rare while urethritis is common. This latter differs from that in the male, however, because of the anatomic contiguity of the urethra to the vagina and cervix and proximity to the vesical trigone. For this reason, urethritis will be discussed in the next section.

Urethral Caruncle is the principal disease of this organ in the female. "Caruncle" is used to name two entities, one a proliferative tumor secondary to irritation and the other a new growth. The difficulty in differentiation lies in the tendency toward inflammation in new growths, but is relatively unimportant. Skene's glands are believed to be the source of these tumors, it has long been observed that these tumors and Skene's glands both lie posteriorly. Another characteristic is the presence of large numbers of dilated capillaries in certain of these caruncles.

The *treatment* consists of infiltration with local anesthetic at the base of the tumor, careful examination to determine its extent, then traction on the tumor and excision at the base with knife or scissors followed by thorough electrocoagulation. Larger sessile tumors had best be actually excised and sectioned for the detection of malignancy.

THE BLADDER

Cystitis. *Cystitis* made up almost half of all the diseases of the bladder seen in this clinic and formed 10 per cent of all urologic diagnoses. The specific forms such as tuberculous cystitis are relatively uncommon, and the usual acute or chronic cystitis has no such specific characteristics as these. The fundamental fact is that inflammation of the bladder is rarely found as an isolated morbid process, it is secondary to disease in the upper or lower tract, or of the adnexae. Consequently, the type of cystitis will vary through fine gradations depending on the source and form of the infecting organism, its duration, and the particular reaction of the parts of the bladder to it.

Cystitis as a diagnostic entity is found much more frequently in women than in men. The reasons for this need only to be mentioned here, for they will be more completely discussed under the various lesions. The two routes for infection in women are through a short, readily traumatized urethra and

through short lymphatics from a cervix which is often infected. In addition, the presence of squamous epithelium almost uniformly in the distal one-third of the urethra and frequently on the trigone as well, which takes part in senile changes with the vaginal mucosa, facilitates infection of this region. A word here about urethritis in the absence of involvement of the trigone or bladder. In general the etiology and bacteriology is the same, and the treatment similar, being the use of gradual dilation with Kollmann dilator to 38 F, irrigation, and if indicated, estrogen treatment as discussed in a later section.

Types of Cystitis Particular forms of cystitis have been differentiated which are based on descriptive differences rather than on actual disease entities.

Proliferative lesions are the result of many types of infection or irritation and are of infinite gradation and mixture. However, four groups may be distinguished.

a *Cystitis granulomatosa* is characterized by fleshy granulations either localized or generalized, and consists of a local inflammatory reaction with disturbance of the epithelium, increased vascularity, round cell infiltration, and increased fibrous tissue. The typical example is seen at the area of contact of an urethral catheter.

b *Cystitis glandularis* is the result of chronic irritation (cases of exstrophy are good examples) effecting metaplasia of the vesical epithelium. There develop acini lined with cells closely resembling the true mucous glands of the small intestine. The relation of these acini to the cysts in cystitis cystica seems to be simply that the acini have sealed over to form cysts.

c *Cystitis follicularis* (papular cystitis) consists of many lymph follicles just beneath the mucosa and is found in cases of chronic inflammation but may persist after the subsidence of symptoms. Colon bacilli are found frequently as etiologic agents.

d *Cystitis cystica* is thought to originate in the downgrowths of epithelium which become cystic either by central degeneration or by cellular secretions. The condition is probably related to cystitis glandularis as already mentioned. It is related to pyelitis and ureteritis cystica as well and is often found concomitantly with these lesions.

Epidermidization embraces several somewhat dissimilar conditions.

a In *cystitis associated with senile vaginal atrophy*, epidermidization has recently come more into prominence by two lines of observation, one that of Cifuentes, and one that of clinical workers such as Everett and Howard.

Cifuentes frequently noted whitish areas on the trigone which differed from normal bladder mucosa (Fig. 142). By biopsy specimens, he was able to compare the microscopic characteristics of this tissue with bladder mucosa and with vaginal epithelium. He found that tissue from these milky areas closely resembled vaginal epithelium both in cellular detail and configuration and in their glycogen content by special stains. He argues that the presence of "flat stratified epithelium of great thickness with clear cells of large size containing glycogen and presenting a layer of intraepithelial cornification, gives us sufficient ground to infer the normal presence of epithelial islands of a vaginal type in the bladder in some adult females." The importance of this work lies in the ability now to differentiate between the true inflammatory trigonitis as a source of cystalgia which will be amenable to treatment, and this "normal" condition of vaginal epithelium on the trigone which is not a

cause of cystalgia, although it may be present with it, and which is not altered by therapeutic measures

This work of Cifuentes opens again the question of the embryologic relation between the epithelium of the vagina and the trigone, both derivatives of the urogenital sinus, and seems to help in our understanding of the action of the female sex hormones on inflammations of the bladder neck. The distal portion at least of the normal urethra has long been known to be lined with squamous (vaginal) epithelium, it stains similarly with iodine. As will be seen in the following discussion urethritis in postmenopausal women is in the same category as cystitis (trigonitis) and for the above basic reason. Everett in 1941 called attention to the relation between the severe cystitis with minimal signs in postmenopausal women and the presence of senile vaginal atrophy, he found that the cystitis responded readily to estrogen therapy. Salmon, Walter, and Geist also in 1941, reported similar results. Howard from this clinic re

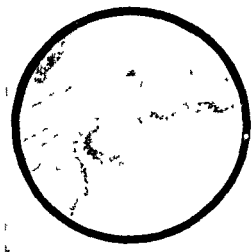


Fig 142 Endoscopic picture of a rather extensive zone of flat stratified epithelium which may be mistaken for a lesion of trigonitis (Cifuentes in *Journal of Urology*, 57, Williams and Wilkins Company)

ported on the treatment with estrogens of conditions complicated by senile vaginal atrophy in ninety-two women and found that stilbestrol applied vaginally was a "valuable adjunct to orthodox therapy in approximately three fourths of the cases"

The criteria for proper selection of the cases are (1) marked urinary symptoms such as frequency, dysuria, and urgency with minimal findings on complete urologic and gynecologic study, (2) senile vaginal atrophy, (3) usually some eversion of urethral mucosa at the meatus with evidence of irritation

The diagnosis of *senile vaginal atrophy* is made by knowledge of the patient's age and history of menopause and by vaginal examination. A valuable adjunct to diagnosis (used routinely in this clinic) is a vaginal smear stained by Mack's iodine vapor method. The technic is (1) dry the smear in air, (2) expose to vapors of fresh Lugol's solution for two to three minutes, (3) grade according to the percentage of cells containing glycogen, those with more stain

being at the more normal end of the scale. An alternative variation is flooding the air-dried smear with Lugol's solution and reading it wet. Not only will this method diagnose the degree of senile atrophy and hence the degree of estrogen deficiency, but it is useful in following the effects of replacement therapy.

The *treatment* is first the correction of all other abnormalities of the urinary tract revealed on complete urologic study, for while senile vaginal atrophy is present in from 40 to 70 per cent of postmenopausal women (Salmon and Frank), it is not necessarily the cause of the inflammation. It may merely occur with sources of irritation and infection and allow them to gain a foothold in the trigonal or urethral epithelium. Restoration of the normal premenopausal health of the epithelium is thought to act by making it more re-

trigone by contamination at the urethral meatus and by bacterial irritation carried to the base of the bladder by the continuity of the cervical and vesical lymphatics (See p. 658.)

Estrogen treatment is best accomplished by the application of a vaginal suppository of 0.1 mg. stilbestrol nightly for twenty-one days, followed by a seven day rest period. In spite of this precaution, a certain number of patients will notice bleeding on withdrawal and should be so warned. Improvement will usually be noted by the end of the second or third week. The course may be repeated from two to four times, when it may be stopped for several months before senile changes and symptoms recur. Occasionally it is necessary to use a natural estrogen if stilbestrol is ineffective, occasionally also, the dosage given causes bleeding on withdrawal, and may be reduced. Vaginal discomfort may occur with the use of the suppository. A trial may be made with stilbestrol ointment or natural estrogen suppositories as a substitute. Oral medication has been found to be less effective and to be accompanied by more untoward reactions (Howard).

b *Pseudomembranous cystitis* (pseudomembranous trigonitis) is more easily classified since the work of Cifuentes. It is the result of a violent exudative process in which the effects of the inflammation are limited to the trigone which becomes covered with a thick fibrinopurulent pseudomembrane. The reason for the restriction of the disease to the trigone can best be explained on the basis of the different embryonic origin from that of the rest of the bladder. Commonly no source for the infection is found and treatment with chemotherapy is found to be fairly effective, especially if combined with urethral dilation and irrigation of the bladder with mild agents. Occasionally the urine is clear, then superficial fulguration may cause the lesion and symptoms to disappear.

c *Leukoplakia* formed 2 per cent of all our cases of cystitis. Of unknown etiology, it may have no pathologic significance. The only known etiologic agent is severe chronic infection. The changes are found in all portions of the urinary tract and consist of thickening and keratinization of the epithelium which resembles skin. These changes, which are usually localized and some-
ia, with actual
tive cornifica-

The *symptoms* are pain and marked vesical intolerance.

Diagnosis is made certain by biopsy specimen, but it must clearly be remembered that in many women squamous epithelium is found normally on the trigone. The significance of leukoplakia has not been satisfactorily explained. Patch felt that the frequent association of the two rare conditions of leukoplakia and squamous cell carcinoma of the bladder (1 in 3 times) indi-

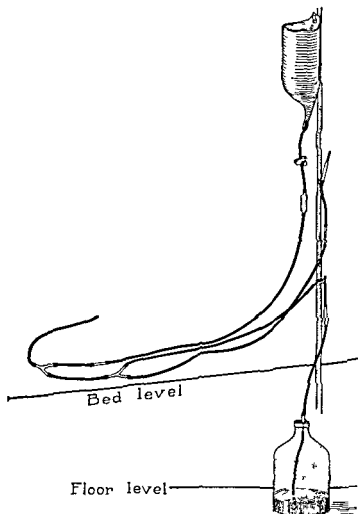


Fig 143 Tidal irrigation. The amount retained by the bladder before the fluid is siphoned off is governed by the height of the tubing going to the bottle on the floor

cate that leukoplakia is a pre-cancerous condition.

associated infection. Superficial fulguration is of some benefit. Fair results have been obtained in personal cases by the use of four or five applications of radium of 100 millicuries well screened directly to the lesions. Vitamin A in high dosage has been used by others with favorable results, although not approaching that in animals with similar lesions.

d. *Incrusted cystitis* appears when the presence of urea-splitting organisms, especially *Proteus vulgaris* and urea-splitting staphylococci, results in extreme alkalinity of the urine, which in turn allows precipitation of calcium, magnesium, and ammonium phosphates as crusts on areas of irritation or

cystoscopic examination reveals complete dissolution, usually a week to ten days (Fig 143) Other acid solutions such as acetic acid or citric acid may be used, as well as half-strength "G" solution if there is intolerance at first to the full-strength material Occasionally cystoscopic removal of parts of the slough and crusts will hasten healing In addition, chemotherapy (sulfadiazine, methenamine, streptomycin) must be given to control the underlying infection, as well as treatment directed at the original disease

e. *Vesical ulcers* may be grouped into (1) simple ulcer, the result of excussion here

Interstitial cystitis is still a disease of unknown etiology, in spite of much recent work by Powell, Barnes, and others Bacteria, toxins, chemical and mechanical irritants have been successively considered without confirmatory evidence being adduced Powell now suggests that obstruction of the intramural lymphatics is the primary lesion

The *pathologic changes* are thinned epithelium resting on a condensed infiltrated submucosa With healing, scar tissue replaces the usual areolar submucosal layer

The *symptoms* are urinary frequency and suprapubic pain, which vary from time to time in severity The pain is intensified greatly by distention of the bladder. The urine may or may not be bloody and is uninfected, and the bladder capacity is small The *diagnosis* is made by cystoscopy with the finding of a short linear area of submucosal discoloration, often with a grayish central line flanked by intense red borders On inadvertent distention of the bladder tiny hemorrhages appear along the axis, and may be enough to obscure vision The ulcers are multiple in half the cases, and are found interconnected over the dome and lateral walls The remainder of the bladder appears normal

The *treatment*, because of the unknown etiology, is not standardized

are often required to cover adequately all the involved area Gradual vesical dilation has proved very beneficial, and recently Powell has advocated suprapubic vesical massage in conjunction with it, insisting that by this means the submucosal lymphatics are opened and normal lymphatic drainage restored

Irradiation reactions should be discussed here because of their confusion with other forms of cystitis and with vesical tumors The *diagnosis* of acute radiation reaction is not difficult, it rests on finding the symptoms of severe

relation to the radiation therapy is not obvious. Frequently they are erroneously considered to be carcinoma. Hematuria, painless or with dysuria, heralds the onset. The cystoscope shows (1) circumscribed areas of telangiectases, or (2) ulceration, and bullous edema in addition, or (3) the above plus a variety of complications acquired during the course of the disease, depending on the severity of the initial insult. The lesion is almost always confined to the posterior wall of the bladder above the trigone (Watson, Herger, and Sauer). The course is usually favorable. Treatment is directed at clearing the specific lesions and symptoms, with cautious coagulation of bleeding vessels, control of secondary infection (use weak silver nitrate solution and oral chemotherapy), and treatment of the complications such as removal of the slough by the Young cystorhynchus, dissolution of calcareous deposits with solution "G," and diversion of the urinary stream in the presence of a urinary fistula.

A contracted bladder was found in 16 per cent of our older patients with bladder disease. Some of these conditions were caused by irradiation, some by prolonged infection, especially tuberculosis, and some by interstitial cystitis. The symptom of frequency approaching incontinence may make transplantation of the ureters necessary (in two of our cases), but a trial of hydraulic dilation should be attempted. A condom or finger cot may be secured to the end of a urethral catheter and used for distention, since incompetent ureteral orifices are found in many of these patients and reflux would occur if direct distention were attempted.

Neoplasms. Second only to cystitis in frequency of occurrence in the older age group are epithelial neoplasms of the bladder (20 per cent of all vesical lesions).

There is little need to be concerned with classification. Ninety per cent are either papillary epithelioma or carcinoma, 5 per cent are scirrhous, squamous or adenocarcinoma, and the remaining 5 per cent rare mesothelial and heterotopic forms.

Papilloma, papillary epithelioma, and papillary carcinoma are the common tumors of the bladder. At this clinic, all papillomata are considered potentially malignant and the pathologic and clinical diagnosis of 'benign papilloma' is avoided. Papillary epithelioma differs from papillary carcinoma only in the absence of infiltration of the base, for it is probable that benign papilloma, papillary epithelioma, and papillary carcinoma are stages in the same disease process.

Pathologically, these papillary tumors consist of many branches of connective tissue covered by columnar or transitional epithelium in one or more layers. Cystic formation, metaplasia, and inflammation do not indicate malignancy. Malignant changes here are similar to those elsewhere and especial weight must be placed on the location of the atypical cells, that is, their tendency to break through the basement membrane and to infiltrate the stroma. They must all be treated as malignant tumors. Recurrence is a characteristic and takes place probably because of continued action of the factor or predisposition which caused the original tumor rather than by implantation. The latter does occur as shown by recurrence in the suprapubic wound after segmental resection. Papillary carcinoma is the common cancer of the bladder, with ulceration and submucosal involvement of the bladder wall in the later stages. Infiltration proceeds through the muscularis and determines the degree of curability by operative means.

Scirrhus carcinoma is a rare form which, because of the more atypical cells, infiltrates rather than forms papillary masses.

Squamous cell carcinoma is of relatively infrequent occurrence and is similar microscopically to the same lesion of the skin. It may be cornifying or not. Often it is difficult to distinguish from other malignant lesions of the bladder, but the characteristic "crunch" on biopsy will give a hint. The probable relation of this carcinoma to leukoplakia has already been discussed. Because of its infiltrating habits it may be missed until ulceration occurs. It is the most malignant and the most radioresistant of bladder tumors.

Adenocarcinoma, as well as sarcoma and other nonepithelial tumors, are

cernible. The symptoms of ureteral obstruction are not rare, due to the paratrigonal sites of occurrence causing partial ureteral occlusion. The cystoscopic picture, while fairly typical, can be interpreted only after considerable experience, for so often it will resemble chronic cystitis with ulceration or edema. Biopsy should always be done, but differentiation must be made between the

factor in the diagnosis is the estimation of the extent and depth of penetration of the tumor, for it is upon this information that the degree of radical surgery necessary for cure will be based. Jewett rightly stresses bimanual abdominorectal palpation under anesthesia as the best means for gauging the extent to which the tumor has penetrated the vesical muscular coats and has fixed itself to the perivesical tissues.

Treatment is based on diagnosis. Simple papillomata are excised with the resectoscope loop followed by thorough coagulation of the base. Follow-up cystoscopic examination must be made in two months and at regular intervals thereafter, for the initial treatment should be considered merely as a therapeutic test for malignancy with the intention to apply more radical measures later if indicated.

Partial cystectomy is performed for those with infiltration, if such excision is technically possible. The distribution of these tumors about the ureteral orifices makes this method applicable only occasionally. Radical cystectomy will more often be the necessary procedure, now done at this clinic in one stage with bilateral ureteral intestinal transplantation. Although operative mortality is not prohibitive, late results with this method for cancer have not been too encouraging. Of course the disease is fairly far advanced before the patient

it then acts only palliatively. Radium is rarely used in these patients at this clinic.

Vesical Calculi. Stone in the bladder is almost exclusively a disease of men

(366 out of 371, July) It occurred in 9 per cent of all our patients with bladder disease. The arbitrary division of these chapters makes it necessary to discuss vesical calculi as if they formed an entity, actually, the whole urinary tract must be considered in each case. Usually the stone arises in the kidneys but would be passed from the bladder were there not mechanical or neurogenic obstructive uropathy of the lower tract. The stone itself acts secondarily as a source of infection and obstruction which are the cardinal factors in the building up of concretions. Cystitis with ulceration by mechanical irritation and vesical trabeculation and cellule formation by vesical neck obstruction are local changes. Obstruction at the bladder neck or by direct pressure on the

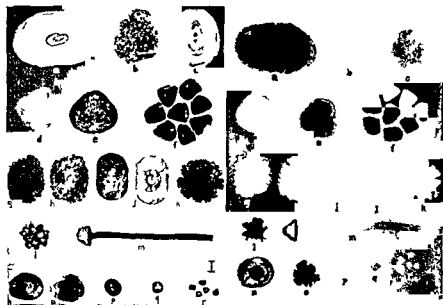


Fig 144 I Collection of vesical calculi a C amount of calcium oxalate c Cross section of b calcium oxalate and phosphate e Cross section phosphate g Calcium oxalate h Cross section calcium oxalate j Cross section of i shell and nucleus examined k and l Calcium oxalate m Calcium phosphate deposits on a mushroom catheter n Calcium phosphate o Calcium oxalate p Uric acid q Calcium oxalate r Calcium oxalate trace of uric acid

II X ray permeability of the stones described above. Note that the calcium oxalate stones are opaque the uric acid stones permeable (b c i j and p) (Hinman Principles and Practice of Urology after Young and Waters)

ureteral orifices leads to bilateral diffuse pyelonephritis. The common causative factors in the production of vesical stones are prostatic enlargement, vesical diverticulum, cord bladder, and urethral stricture.

Diagnosis is not difficult. The symptoms are as would be expected: those of infection (cystitis and pyelonephritis) and obstruction. Sudden interruption of the urinary stream is a classical but not necessarily pathognomonic symptom. The x ray gives strong presumptive evidence (Fig 144) especially if oblique views are made. Confirmation can be had by air or skiodan cystograms which include an oblique view. But determination of the presence of the stone is, of course, not enough; the etiologic disorder in its formation

must be found. Work up will include measurement of residual urine, rectal palpation, and films of the upper tract often with intravenous dye. Cystoscopy is usually indicated because the stone may be associated with papillary tumors of the vesical wall, in order to visualize orifices of associated diverticula, and to study the vesical neck and prostatic urethra for obstruction. Vesical atony and trabeculation are also noted. An accurate estimation of the size of the stone and of its consistency is important, for the choice of the method for removal will be based on these observations.

Treatment The treatment hinges on the general condition of the patient and the nature of the obstruction leading to the formation of the stone. *Dissolution* the noninstrumental method, is slow and uncertain and should be reserved for those with associated disease which contraindicates manipulation or operation. Suby's solution "G" (sodium carbonate 4.4 gm., citric acid 32.4 gm., magnesium oxide 3.8 gm., distilled water 1000 cc.) is used full strength, if tolerated, through an urethral catheter. A tidal irrigation apparatus is advisable so arranged that some distention of the bladder occurs with each filling. Too often, however, a stage in the dissolution is reached in which the stone is left covered with an insoluble organic layer. In spite of the promising work of Keyser and others with enzymes further solution of the stones is difficult. An oxalate stone (unusual in the bladder) likewise is not soluble in weak acids.

The choice of the *instrumental method* depends on the nature of the underlying obstruction. First, the stone may be removed incidentally to prostatectomy or diverticulectomy. Secondly, litholapaxy may be indicated. Lastly, (and only if the other two possibilities are inapplicable) suprapubic cystolithotomy may be done.

(1) Removal of the stone for an associated condition needs little additional explanation. If an obstructing prostate is involved, the size of the stone may have a bearing on the choice of procedure (transurethral, perineal, or suprapubic prostatectomy).

(2) *Litholapaxy* is the safest method. It is not applicable when the stone (a) is associated with a condition necessitating operation or prohibiting passage and manipulation of a lithotrite, (b) is too large to be grasped (approximately 4 to 5 cm.) or (c) is formed about a foreign body. The operation is, however, difficult to perform and should be left for the trained urologist. Preparation is as for any transurethral operation. Probably the best combination is the use of the Bigelow lithotrite, followed by evacuation and inspection through a resectoscope sheath. Larger fragments which may remain may be crushed or withdrawn with Young's cystoscopic lithotrite.

(3) *Open operation* is now performed exclusively suprapubically, often under local anesthesia because of the age and debilitation of the patient. A high vesical incision is desirable since it is followed by earlier closure of the cystostomy fistula. Often open cystolithotomy is combined with other procedures intended to relieve vesical neck obstruction, remove diverticula, and the like.

Vesical Diverticulum As would be expected, diverticula of the bladder are relatively common in men over sixty (5.2 per cent of all vesical disease in the present series).

Three types of diverticulum may be found. (1) True diverticulum with sacculation of all layers muscular and mucosal. (2) true diverticulum, with

herniation of the mucosa alone through a defect in the musculature, and (3) false diverticulum, after perforation or rupture, in which the cavity is not lined by vesical elements. To be differentiated from these are double or hour glass bladder, dilated urachus, and rudimentary ureteral buds. The last are rare and are not in the province of geriatric urology (only one of our thirty four cases was congenital), but a word may be said about dilated urachus. Not infrequently, a patient with obstruction at the vesical neck is seen with mild vesical decompensation with residual urine. If a cystogram is made before the pre-operative catheter drainage allows the hypertrophied detrusor to tighten down, a conical outpouching is found in the midline superiorly which represents a dilation at the site of the obliterated urachus. This is of no clinical significance.

The constant *etiological factor* of diverticulum is obstruction at the vesical outlet. It is to be expected, then, that diverticula are thirty times more common in men than in women (18 female to 543 male, Schacht and Crenshaw). There are three components necessary to their formation. (1) The *anatomic factor* is that since the detrusor consists of three poorly defined interlacing layers, hypertrophy results in thinning of the internal layer and local enlargement of individual muscle bundles of the middle and external coats. This is especially marked in the region just outside the trigone for here the structures are fixed during urination and there results the greatest strain upon the coats. It is well known that trabeculation and cellule formation are seen first in this region, yet this region histologically is thicker than other portions of the bladder. For this reason, herniations are ascribed to functional strain rather than anatomic weakness, for over 90 per cent of diverticula occur about the trigone. (2) The *pathologic factor* is concerned with specific weaknesses of the components of the vesical wall. These changes occur predominantly with age (atrophy of the muscle fibers with increase in the interstitial connective tissue and in the elastic fibers) but inflammatory processes are often superimposed to increase the amount of relatively weak connective tissue. (3) The *mechanical factor* operates through long continued obstruction at the vesical neck rather than by an acute obstruction. In the gap between the compensating (hypertrophic) muscle bundles, a cellule is formed and two additional factors cause its growth. First, the hypertrophied bundles act as a sphincter to close the diverticular orifice and cause the thinned out detrusor fibers on the diverticular wall to contract against a sphincter. Alternately, these bands may cause the orifice to stay open, thereby allowing the full force of intravesical pressure to act on the weakened diverticular wall.

The *diagnosis* is rarely made by pathognomonic symptoms (necessity for emptying bladder twice, the second time often in a particular position) but in investigations for infection and obstruction will suggest its presence. In older men it will almost always be associated with some form of vesical neck obstruction, and the question will arise as to whether relief of this block will be
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especially if associated with stone or tumor, should be removed. Better drainage and removal of stone from the diverticulum may sometimes be accomplished by judicious resection of the diverticular orifice transurethrally. For

(junger) men,

open operation adequate exposure by the Cherney incision with combined intra and extravescical excision will most often give the best results. It must be remembered that the operative mortality for diverticulectomy is much higher than for prostatectomy. For this reason vesical diverticulum in the aged is usually treated medically by urinary antiseptics and periodic bladder irrigation through a catheter after relief of the causative obstruction.

Vesical Fistulae Fistulae from the bladder accounted for 2.6 per cent of all bladder disease in our series. They are of three varieties, all of which are usually secondary to some surgical procedure.

Vesicocutaneous fistulae are almost always secondary to perineal or suprapubic surgery and both tend to close spontaneously when vesical neck or urethral obstruction is eliminated. *Vesicointestinal fistulae* are not rare, those to the rectum being most common (in men from perineal surgery, less often in women from pelvic surgery). *Vesicovaginal fistulae* are a common result of accidental injury at operation or during parturition.



Fig. 145. Cystograms illustrating the value of a contrast picture. A. Cystogram which fails to demonstrate a diverticulum. The contrast cystogram B demonstrates two, one fairly large on the left and one small one on the right. (Herman: Principles and Practice of Urology.)

The *diagnosis* of urinary fistula is often based first on finding an uriniferous drainage. There may be pneumaturia and fecal drainage in the urine in intestinal fistulae, which is confirmed by cystograms and barium enema. At times the fistula may be felt per rectum. The orifice in vesicovaginal fistula may be seen directly or located by packing the vagina and instilling methylene blue into the bladder.

The *treatment* is first directed to spontaneous healing by institution of drainage either by urethral catheter or cystostomy. If the fistula is due to malignant disease, diversion of the fecal stream will often allow healing, or at times

vesicovaginal fistulae is often a difficult technical feat, and many of these patients have had two and three attempts. Usually the vaginal approach with vesical atony and parastomal hernia and formed 2.1 per cent of all bladder disease in our series. Because of the confusion in classifica-

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The *diagnosis* is rarely made by pathognomonic symptoms (necessity for emptying bladder twice the second time often in a particular position) but investigations for infection and obstruction will suggest its presence. In older men it will almost always be associated with some form of vesical neck obstruction, and the question will arise as to whether relief of this block will be enough to prevent prolongation of urosepsis or whether it must be combined with or be followed by diverticulectomy. Contrast cystography (Fig. 145) and cystoscopy will establish the diagnosis.

Treatment is often expectant. Large diverticula in healthy (younger) men, especially if associated with stone or tumor, should be removed. Better drainage and removal of stone from the diverticulum may sometimes be accomplished by judicious resection of the diverticular orifice transurethrally. For

open operation, adequate exposure by the Cherney incision with combined intra- and extravescical excision will most often give the best results. It must be remembered that the operative mortality for diverticulectomy is much higher than for prostatectomy. For this reason, vesical diverticulum in the aged is usually treated medically by urinary antiseptics and periodic bladder irrigation through a catheter after relief of the causative obstruction.

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The *treatment* is first directed to spontaneous healing by institution of drainage either by urethral catheter or cystostomy. If the fistula is due to malignant disease, diversion of the fecal stream will often allow healing, or at least alleviate the symptoms. In uncomplicated cases, excision of the opening and closure is to be tried, in more difficult cases of rectourethral fistula a modified Whitehead operation (Young and Stone) is indicated. The closure of

tion, we have included a brief discussion of the nervous mechanisms of urination and their relation to disturbances of emptying (See p 288)

Normal urination consists of, first, cerebral release of inhibition, especially by relaxation of the resting tone of the external sphincter, then contraction of the detrusor of which "relaxation of the internal sphincter" (an appendage of the detrusor) is an integral part. This in turn causes reflex relaxation of the external sphincter. At the termination of urination, contraction of the external sphincter and muscles of the bulb and perineum completes the act.

The *nerve supply* to the bladder is from three sources. The *parasympathetic* is the most important and constitutes the main reflex arc. Its stimulation contracts the bladder by activation of the detrusor. It arises from S2 and S3 via the pelvic nerves and hypogastric plexus. Isolated section first causes inability to contract the detrusor and empty the bladder. Later, some degree of automaticity obtains by the inefficient intrinsic innervation of the muscle, and hypertrophy ensues. Vesical tone and function is dependent principally upon the intact parasympathetic reflex arc in the conus. Disturbance of the afferent limb causes vesical atony, the commonest type of neurogenic bladder in old age. The *sympathetic* nerve supply, which is relatively unimportant and may be disregarded for practical purposes, arises from L1 and L2 through the thoracolumbar outflow. Its stimulation causes a short rise of pressure, then a short fall below normal. It pulls down the ureteral orifices, and in males increases urethral resistance. Section causes no change in vesical emptying. The *somatic* supply enters with the pudendal nerve from S3 and S4. Stimulation causes closure of the external sphincter (striated), while section causes no change in urination if the internal sphincter is present. It must be noted that the external sphincter is innervated from both parasympathetic and somatic sources.

The syndromes possible from derangement of these mechanisms are four.

The *atonic neurogenic bladder* is caused by loss of the sensory reflex arc, typically by the syphilitic damage in tabes dorsalis, but also in combined system disease, diabetes, multiple sclerosis, and amyotrophic lateral sclerosis, in which decreased sensation to filling permits distention. This results in poor detrusor tone with fine trabeculation and funnel orifice as the cystoscopic findings, and in overflow incontinence as the principal symptom. The treatment is the avoidance of overfilling in early cases. Adequate drainage may allow some tone to return to the muscle. Frequently the surgical removal of an enlarged prostate, median bar, or even of a portion of a normal vesical neck will decrease ureteral resistance sufficiently to allow the weakened detrusor to empty the bladder.

The *autonomous neurogenic bladder* is secondary to loss of both afferent and efferent parasympathetic arcs, usually from injury of the cauda equina.

Urination occurs involuntarily in dribbles, closely resembling true overflow incontinence. Treatment is directed primarily at diminution of urethral resistance by *transurethral resection*. Several resections, with removal of relatively large amounts of tissue, are often necessary to allow more complete emptying but frequently, because of the inefficient reflex, incontinence con-

tinues. Higher cord section has recently been advocated for the more hopeless of these cases, to transform the bladder into a reflex neurogenic one (see below) with its more automatic function. The theoretic objection to this is that half the low reflex arc is already blocked and a desirable degree of automaticity may not develop. Abdominal compression should be tried and may be an aid to more complete emptying. Too often, treatment can be only palliative requiring suprapubic or urethral catheters.

The *reflex (automatic) neurogenic bladder* is caused by high cord section (above S1 although some degree of automaticity may persist with lower lesions) such that the lower reflex arcs are left intact, and only cerebral initiation and inhibition are cut off. Spinal cord injury causes first a stage of shock in which normal tone alone is the rule, then a state of automaticity with absence of sensation of filling, a small residual (and often small capacity), and inability to initiate micturition. Often there is some warning of impending urination. Skin stimuli (sacral distribution for bladder and caudal area for external sphincter) may initiate reflex urination. The treatment consists of (1) tidal irrigation until automaticity begins, (2) removal of irritative stimuli such as stones and infection, and (3) training to void at definite intervals. Abdominal compression may help. Resection of tissue at the vesical neck may be indicated if residual urine persists.

The *uninhibited neurogenic bladder* is due to loss of central control by a cerebral lesion (hemiplegia, tumor, multiple sclerosis). These patients have frequent urgent urination because the parasympathetic stimuli to the bladder are not inhibited by the higher centers. Atropine or ephedrine (the one by opposing parasympathetic action, the other by increasing sympathetic function) may be tried for treatment.

Another form of nervous disturbance in old men may be discussed here. In *parkinsonism*, the striated external sphincter takes part in the general increased muscular tone. This resistant sphincter is associated with a small strong bladder. Since a poorly relaxed sphincter will give symptoms of prostatism, care must be taken in evaluating prostatic obstruction in men with Parkinson's syndrome.

Another disturbance not strictly neurologic is *incontinence* in men and women. In men, trauma to the external sphincter at prostatectomy (especially transurethral resection and perineal prostatectomy) accounts for most cases. Perineal surgery (prostatectomy or combined abdominal resection of the rectum) may interfere with the nerve supply to the sphincter. Incontinence results. More often after the latter procedure, retention follows due either to interference with vesical innervation or to posterior displacement of the bladder by withdrawal of its normal supports and consequent angulation at the outlet. This may be diagnosed by finding that the patient is able to void if he presses on the perineum or if he gets on his hands and knees. The treatment may be by a special perineal support or by operation. However, often in post-operative retention a period of tidal irrigation will allow the bladder to regain tone.

In all forms of neurogenic bladder disturbance in the elderly male, the high incidence of complicating mechanically obstructive lesions of the prostate and vesical neck must be borne in mind. When the vesical innervation is damaged, the bladder is much less able to compensate for obstruction than when its nerve supply is intact. For example, a mild degree of vesical atony

tion we have included a brief discussion of the nervous mechanisms of urination and their relation to disturbances of emptying (See p 288)

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prostate, median bar, or even of a portion of a normal vesical neck will decrease ureteral resistance sufficiently to allow the weakened detrusor to empty the bladder.

The *autonomous neurogenic bladder* is secondary to loss of both afferent and efferent parasympathetic arcs, usually from injury of the cauda equina. The coarse trabeculation found at cystoscopy is due to the fact that the short reflex arc intrinsic in the detrusor keeps acting in an inefficient manner. There is retention at first, then some weak reflex urination with residual urine. Often urination occurs involuntarily in dribbles, closely resembling true overflow incontinence. Treatment is directed primarily at diminution of urethral resistance by transurethral resection. Several resections, with removal of relatively large amounts of tissue, are often necessary to allow more complete emptying but frequently, because of the inefficient reflex, incontinence con

"hang" must not necessarily be interpreted as due to stricture, for spasm may give a similar sensation

Treatment is directed first at removal of the cause, rather than the effect, for removal of extrinsic obstructions and intrinsic strictures will be much more effective than treatment of secondary dilation and angulation. Dilation over a long period of time may be necessary, but more often direct surgical attack will give better results. A trial of dilation may be advisable but should not be persisted in if improvement is not demonstrated. Numerous plastic procedures have been described for release of these strictures, but at the present time the intubated ureterostomy (Davis) is the most popular. In this technic, a relatively large soft rubber catheter is passed through a nephrostomy incision down the ureter past the stricture which has been incised longitudinally. A few loose fine catgut sutures may be used to tack the strip of ureter thus formed to the catheter. The tube is removed in from two to three weeks, at a time when ureterograms demonstrate no leakage from the incised area.

Ureteral Calculus. Stone in the ureter is a relatively frequent source of ureteral disease in the older age group (one third of our series)

Very nearly all ureteral stones originate in the kidney. The majority pass unhindered to the outside, which explains why less than 5 per cent of ureteral stones are discovered concomitantly with renal calculi. About twice as many occur in men as in women and they are unusual in children. Impaction of a stone in the ureter causes renal obstruction and infection, manifested by fulminating pyelonephritis with septicemia, or more often by varying degrees of infected hydronephrosis. Of course before descending into the ureter it may have caused direct damage to the kidney.

The early *symptoms* of ureteral stone are well known, with the referred pain more or less characteristic for the three portions of the ureter. Small stones are more likely to produce the most pain. The later symptoms are those of obstruction and infection of the kidney. Occasionally very large stones are found, which would indicate the absence of much obstruction for they could not grow without a good flow of urine.

A presumptive *diagnosis* is made by finding blood, and often pus and organisms, in the urine and by a shadow on the plain film in a characteristic position. Confirmation is had by retrograde catheterization but often intravenous pyelograms will be diagnostic. Oblique views frequently are conclusive in placing a suspected shadow within or outside the ureter. However, associated changes of the lower tract (etiologic) and of the upper tract (secondary) must be sought out. In about 10 per cent, the calculus is radiotranslucent and will be located by finding a filling defect in the pyelogram, finding an opacity by adsorption of dye following the pyelogram or detecting scratches on a wax-bulb catheter.

Treatment. The treatment of ureteral calculus is expectant, manipulative, or surgical. The factors determining the choice of treatment are many (the size, shape, and position of the stone, the duration of its presence in this position, the presence or absence of obstruction or infection, the condition of the opposite kidney, the general condition of the patient, and the presence of other upper tract disorders requiring surgical treatment), and require much judgment for the proper course.

The *expectant* treatment utilizes a large fluid output with analgesics and antispasmodics (morphine and atropine are still among the best medications

for this purpose) Periodic observation with excretory urograms are necessary At least 80 per cent of all ureteral calculi will be passed spontaneously if one waits long enough However, it is imperative to know that the affected kidney is functioning satisfactorily and not undergoing hydronephrotic destruction If evidence is found of progressive hydronephrosis or infection, ureteral catheterization or surgery must be resorted to

The *manipulative* method is best limited to stones less than 1 cm in diameter in the upper ureter and less than 2 cm in the lower ureter, which are not fixed and are not associated with serious renal infection The failure of initial attempts or severe reactions following them would make open operation a better course Several methods may be tried One of the simplest is the method of Elhk in which a threaded catheter is passed to the renal pelvis, a loop formed in its tip, and the stone engaged and withdrawn Multiple catheters when twisted may enmesh the stone A Johnson basket is effective in the ureter below the pelvic brim but should not be used above that level Injection of 4 per cent novocain or dilute avertin sometimes appears to have a relaxing effect on the ureter The passage of several catheters, leaving them in twenty four hours, frequently dilates the ureter enough for the stone to pass Meatotomy at the ureteral orifice may be indicated

The *surgical* treatment consists of an extraperitoneal flank incision over the stone for those in the middle and upper thirds, and an oblique lower quadrant incision for those in the lower third, which incidentally are more difficult to recover It is advisable to place a rubber tape above the stone while freeing the ureter so that the stone will not slip back into the renal pelvis The longitudinal incision in the ureter is closed loosely with fine catgut or left open, and adequate drainage provided to the area of the incision Ureterolithotomy is frequently better borne by the patient than manipulation, particularly when the stone is large In fact, the postoperative course is so benign after lumbar ureterolithotomy that surgical removal of the stone is nearly always the treatment of choice when a stone is found arrested above the pelvic brim (Foley *et al*) When the stone is in the pelvic ureter, operation may be difficult In general, the closer the stone is to the ureteral orifice, the more is the likelihood that it may be successfully removed by manipulation

Ureteral Neoplasm These tumors are felt to be very rare but we have seen four cases in patients over sixty years of age in the past ten years *Etiologically* and *pathologically* they are closely related to bladder and renal pelvic tumors The principal forms are papilloma and papillary epithelioma

The *diagnosis* is begun usually with the finding of hematuria In one of our cases, vesical tumors were treated for over a year before the ureteral neoplasm was discovered Retrograde pyelography must usually be resorted to, and will show a dilated ureter and pelvis above a filling defect in the ureter

Treatment is ureteronephrectomy, which usually must be done even for benign tumors because their differentiation preoperatively is difficult and there is so often marked hydronephrosis that repair of the ureter is not indicated

CHAPTER 40

DISEASES OF THE PROSTATE

PERCY S. PELOUZE*

THE prostate, like all other body structures, undergoes changes resulting both from advancing age *per se* and from disease. These are distinguished with difficulty. The gland frequently, but not invariably, increases in size in later years. The tendency to consider pathologic lesions as typical of certain age phases has been productive of great harm. We have called the middle fifties "the prostatic age" for so long that many have overlooked entirely what such a statement really means, and have neglected to consider the prostate seriously during the forties. While it is perfectly true that that is the average time at which most diagnoses of prostatic hypertrophy are made, the clinical picture defies such a fixed belief. Many cases of this lesion are so far advanced when the diagnosis is finally made as to prove that the groundwork was laid down at a much earlier period and that we might do far better, if we must use figures, to call forty-five years the prostatic age and fifty-five years the age wherein other organs more commonly rebel against the *gradually increasing obstructive insults*.

Infections of the prostate, other than gonorrhea, are said to occur at an average of about forty-nine years. But to consider focal infective prostatitis as predominantly a disease of older men is to overlook some very definite reasons why the diagnosis so commonly is not made in the earlier years of life. Almost invariably, the infection has been present for years before it is discovered, and, while it is a very common lesion of later life, it unquestionably is just as frequent between the ages of twenty-five and forty years when comparatively few prostates are studied. Furthermore, to consider tuberculous involvement of this gland as belonging to early life is to overlook the fact that it may occur at almost any age.

As far as prostatic carcinoma is concerned, there is much more reason why it could be viewed as a lesion belonging almost entirely to that period of life with which this volume is concerned. This, however, does not imply that it cannot develop in earlier years.

In recent years our careful internists have urged that palpation of the prostate gland should be included in every study of the adult male. No one would take exception to this teaching as far as it goes. The room for criticism lies in the fact that it does not go far enough, for more lesions are overlooked than are discovered by mere palpation of this gland. Countless patients have their prostates acquitted who should not have and a goodly number are thrown into near panic because of misinterpretations that have little need to occur.

* Deceased

GENERAL DIAGNOSTIC CONSIDERATIONS

Size Perhaps the most frequent errors in prostatic diagnosis are due to faulty interpretations of size. For so many years the size and shape of this gland has been likened unto that of the horse chestnut that the physician's standard of normal is the last horse chestnut he remembers having seen. He loses sight of the fact that horse chestnuts, although of about the same shape, are not always of the same size. As a result, he bases his opinion on a standard that is variable and, when he finds something larger than he expects, he calls it either hypertrophy or congestion if it is soft, or carcinoma if it proves to be of firm consistency.

Unlike other organs, the prostate is normally larger in those more than fifty, often even twice its normal size at twenty years of age. It usually is larger in large heavy-boned men and it commonly is so in their smaller brothers. Because of its variations in shape, it often is extremely difficult to estimate its real size and, as will be pointed out later, it may be, and commonly is, obstructive without any rectal evidence that such is the case.

Shape. Almost as many normal and abnormal prostates do not resemble a horse chestnut in shape as do. Most of these variants are flat on their posterior surfaces, have a deep superior notch, and extend superolaterally.

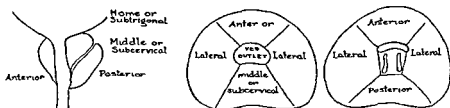


Fig. 146 The locations of the various prostatic lobes and the subtrigonal glands (Pelouze Office Urology)

well up under the seminal vesicles. Medially such glands have but a thin layer of prostatic substance between the urethra and the prostatic capsule. This is particularly evident at the level of the vesical outlet.

Consistency While most normal and infected prostates are of moderately soft consistency, it is by no means rare to encounter individuals in whom the gland is normally almost as firm as is the case with some carcinomatous prostates. This is even more common in the presence of long standing infection.

Nodules Though some few nodulations of the prostate are of inflammatory origin, by far the large majority are due to either carcinoma or tuberculosis. So true is this that no gland presenting such irregularities of outline should be subjected to so-called massage until the closest study and a prolonged period of observation rule out these graver lesions.

Prostatic hypertrophy, when it is discoverable per rectum, is smooth, soft, and not in any sense nodulous. If it presents areas of definite hardness these should arouse suspicion of carcinomatous involvement.

Palpation Alone in the Discovery of Prostatic Pathology. That prostatic palpation alone carries with it a large margin of error in diagnosis has been ably shown by the work of Randall.¹ Unquestionably there is need for a higher level of suspicion regarding such lesions than now holds. The fact that

Randall's cases were studied in a large general hospital upon whose staff are some of the country's outstanding clinicians proves this. A depressing proportion of his cases reached the autopsy table without the slightest suspicion having been raised regarding the role of the prostate gland in the cause of death. The majority of these lesions could not have been determined by rectal palpation alone.

Age and Prostatic Obstruction. Reverting again to Randall's work we find therein reported the occurrence of prostatic hypertrophy as to age groups as follows: 2.2 per cent between the ages of twenty and thirty-nine; 10.3 per cent between forty and forty-nine; and the remainder in those more than fifty years of age. Here can be seen the dangers of linking the questions of age and this lesion too closely in our minds. Certainly there is great need for a wider use of the cystoscope in the study of such lesions than at present holds, and he who too confidently trusts his index finger courts many unpleasant surprises. Such casual methods have caused a world of morbidity and no small number of deaths. Obviously there is an urgent need that the lessons so sadly learned by the urologists should be made known to those in general practice. For it is an outstanding fact that the former still see many patients who have been treated for months, often years, for something else—cases in which the mind of the merest urologic tyro would at once have turned to the prostate gland.

Types of Obstruction. The obstructive prostate may be of several types. Benign hypertrophy and median bar formations lead to obstruction. Inflammations, prostatic calculi, cysts, and fibromyomata or leiomyomata are not infrequently causative of obstruction. Prostatic carcinoma is a less frequent cause, unless complicated by other factors enumerated above.

Symptoms. If one reviews the symptoms experienced by many of those in whom the prostate gland is the site of one or another kind of pathology he finds many reasons why a large proportion of such cases escape diagnosis for weeks—sometimes for years. With but few exceptions these are the symptoms of many patients having no disease in the gland. Careful history taking frequently will reveal ample personality reasons why some of these symptoms may be present, but even that does not prove that some slowly develop and to m wholly a

jective symptoms directing attention to the prostate. Therefore, even minor deviations from the normal deserve close attention.

Frequency of Urination. Probably no one passes through life without one or more attacks of increased frequency of urination. For one reason or another, a fair number of perfectly normal persons have it all their lives. Among these latter are to be found those individuals by no means small in number who, by virtue of an assortment of faulty ideas about the urinary bladder, develop the habit of emptying that viscus just as soon as they are conscious that it contains even a small quantity of urine. Never is the bladder allowed to distend to its full capacity during waking hours, and seldom do such persons avoid developing what has been termed an alarm-clock bladder, which causes them to awake at rather definitely fixed hours during the night. So common is this condition that it gives point to careful history taking in every case to avoid attributing to the changes of age conditions that have

been present during many of the preceding years. These victims of habit are made far more miserable than others when a true obstructive uropathy does set in.

As far as nocturnal frequency in those of advanced years is concerned one does well to leave some latitude for the fact that the aging bladder is not always so comfortably distensible as it once was. This, alone, often causes men past fifty years of age to empty the bladder once or twice during the night. Added to this may be the lessened power of urinary concentration by the kidneys, so that gradually increasing amounts of urine are poured into the bladder during the night. These, together or independently, often supply the reason for the nocturnal urinations of the aged in the entire absence of vesical outlet obstruction and residual urine. One must not assume that these are the sole reasons, however, until the graver conditions have been ruled out.

Residual Urine For so many years the presence of more than a few cubic centimeters of urine in the bladder after urination has been considered one of the indications for operations upon the prostate gland that the subject is well worth discussion. Our great fault in this regard is always to assume that such quantities of residual urine are there because the patient cannot completely empty his bladder because of obstruction. However, there are many who cannot completely evacuate the bladder for other reasons. Frequently the reason is psychogenic, some men are unable to void normally upon demand or in the presence of others. A residual of 1 to 6 ounces or even more warrants investigation, but not immediate diagnosis of prostate bladder neck obstruction. Even in the presence of truly obstructive lesions, wide variations in the volume of urine retained will be observed at different times in the same patient.

Thus, the mere presence of urine in the bladder immediately after urination loses much of its supposed pathognomonic value. Like most other symptoms it needs bolstering with other findings if one would avoid some dramatic errors in patient guidance and, often, in operative procedure. About the only thing that can be said is that *persistent* quantities of *residual urine* in any patient who is alone when he urinates mean either a neurogenic bladder or a true vesical outlet obstruction. Upon rare occasions a hydronephrosis or a vesical diverticulum may be the reason. The latter, however, is rather rare in the absence of obstruction.

Pain As the prostate gland is singularly free of intrinsic pain, such pains as do occur usually arise in other structures, most commonly in the bladder and along the urethra. As is stated under Prostatic Carcinoma, the pains caused by this lesion almost invariably are those due to metastases or vesical outlet obstruction. Minor lesions of this gland most commonly cause trigonal discomfort or referred discomforts along the urethra, into the perineum or the rectum—into the latter particularly in the presence of lesions in the sinus pularis. Intrinsic pain is rarely an outstanding feature even in abscess of the gland. It has not been my experience that the low back pain so commonly considered a symptom of prostatic infections is due to such infection in any significant number of cases if indeed it ever is.

Difficulty in Urination One could discourse at great length upon this in-
 would have to
 , in the entire
 mpt relaxation

of those delicately balanced sphincters that make it possible to hold urine in the bladder. He would have to call attention to the fact that aging commonly makes these sphincters respond less readily to mental stimuli although in childhood they worked with almost lightning like speed. He would have to guide the present-day mind away from that old idea that urethral stricture is a disease of early adult life and call attention to the fact that we have stopped causing strictures by our treatment of gonorrhea, and that those we have with us today are to be found in the older veterans of a not altogether forgotten traumatic past. He would have to call attention to the facts that neurogenic and prostatic lesions commonly cause the symptom but that patients with enormous lateral lobe prostatic hypertrophy may not even have it, and, then, he would have to admit that without careful study one does not know which is which.

INFECTION

It was only when the urologist succeeded in disentangling himself from his former fixed belief that he who had an infection of this gland and denied having had a previous gonorrhea was prevaricating, that the real truth about such infections began to be revealed. From this point on, careful clinicians abandoned the idea that anything short of a microscopic study of the gland's secretion could answer the question of infection. As they did this they soon discovered that infections of the prostate did not belong alone to the earlier years of adult life. If anything, they found that the chronic infections were even more common in later life than in those earlier years wherein they so frequently cause local symptoms directing attention to their presence. Thus did the condition become an eminently appropriate one for inclusion in a work on geriatrics.

Etiology. Contrary to our older views upon the matter, gonorrhea accounts for but few prolonged prostatic infections. In fact, the gonorrheal prostate, unless the gland has been traumatized during the acute stages of its infection or there has been true abscess formation in it, almost always becomes infection free in from two to five years, whether it is treated or not. Hence, gonorrhea very rarely is an etiologic factor in the disease even in midlife and, practically, it does not enter the picture after that period.

At least 95 per cent of older individuals with prostatic infection have either infected tonsils, tooth-root abscesses, or infected gum pockets. To make the etiologic tie up more complete, we find that it is decidedly unusual for us to be able to render the gland permanently free of infection until these more distant foci have been removed. Furthermore, recurrences of infection in such glands as have responded to treatment after the removal of distant foci almost always are preceded by recurrent tooth infections. So true is this that one very appropriately can consider the focal infective triad as being teeth, tonsils, and prostate gland.

Beyond this, there unquestionably are a few prostatic infections that are secondary to such conditions as upper urinary tract, intestinal, and gallbladder infections, influenza, and probably other systemic infections.

Symptoms. Except for the acute infections of the prostate gland, which are rare except in the presence of gonorrhea, it is extremely uncommon that there be any sensory symptoms in the gland itself. Before forty-five years of age the most common symptom suggesting the possibility of such an infection

is nonspecific urethral discharge. After that age, urethral discharge is very rare and the condition usually is discovered during studies to determine the causes of distant symptoms suspected of being focal-infective in origin. In a few cases the prostate is studied because of a pyuria found not to be due to upper urinary tract infection.

Upon the rarest occasions an added congestion gives a feeling of fullness in the region of the gland. Slightly more often there is a feeling of fullness in the rectum. All in all, the infection is practically silent locally and is discovered during an attempt to explain symptoms in contiguous or distant structures.

Clinical Importance. Aside from the clinical importance of prostatic infections as a cause of pyuria discovered microscopically in the urine or as the occasional cause of secondary cystitis, they assume a far wider importance in their role as foci of infection causing distant symptoms in the joints, the eyes, or in other structures. As the arthritides are more common in later years, a search for such prostatic foci is a matter of considerable importance in older men (see p. 678).

No matter what view one may take of limited foci of infection as factors in the causation of many of these systemic or local reactions, prostatic infections are still of importance in the general picture and the more one studies them the more convinced he will be of their frequent association with these systemic or local conditions. Not that such a prostatic infection, of itself, precipitates these distant symptoms, but that not infrequently it is the sole cause of their continuance after all other things have received attention. The truth of this is easy of proof in many cases, and in those cases wherein absorption from the prostatic infection is not a factor, that, likewise, is easily determined. In approximately 50 per cent of those cases that do cause absorptive symptoms the patient becomes symptomatically worse within twelve hours of any digital disturbance of the gland and his increase in symptoms usually subsides within twenty-four hours. Occasionally, particularly in the presence of active arthritis, this symptomatic reaction lasts for several days. These reactions occur in practically every case of focal infective eye condition wherein prostatic infection is a factor and they often are matters of grave concern. So fixed are these reactions in these two latter conditions and in markedly active arthritis that the absence of such exacerbation is excellent proof that the prostatic infection is playing no role.

In those arthritic and neuritic cases wherein digital pressure on the gland gives no distant reaction, it is only safe to say that the prostatic infection is not a factor if repeated prostatic massages fail to exercise any beneficial influence on these distant symptoms.

The question of the reputed etiologic role of prostatic infections in prostatic hypertrophy will be discussed under the subject of Hypertrophy.

Diagnosis. The diagnosis of prostatic infection is entirely dependent upon the microscopic study of the secretion of the gland. The normal prostatic secretion frequently contains as many as six polymorphonuclear leukocytes to the high-power field. Therefore it is held that any number in excess of this is an evidence of infection in the gland. In the presence of infection deep in the gland the number of leukocytes may be as high as 100 or one heavily infected leukocyte may be seen. The number of leukocytes is occasionally, but not always, high and these are true

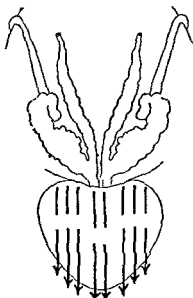


Fig 147 Direction of the stroking of the prostate gland. By leaving the midline strokes until last the discomfort is reduced to a minimum and is at the end of the treatment (Pelouze Office Urology)

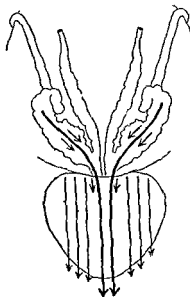


Fig 148 Prostatic massage and stripping of the seminal vesicles (Pelouze Office Urology)

evidences of infection and not caused by the repetition of the digital manipulations. Repeated massage of a normal gland does not cause an outpouring of leukocytes

Treatment. It is rare, indeed, that the administration of any of the *sulfonamide drugs* exercises a beneficial effect upon nongonorrheal infections of this gland, this despite the fact that most of them are due to bacteria, staphylococci, in which one of these drugs, sulfathiazole, is particularly valuable. *Penicillin*, to be effective, must be administered in liberal dosage.

Massage There is a right way and a wrong way, as well as a right time and wrong time for massage. It should be remembered that it is used for the treatment of infection, that this is about the only structure in the body wherein such measures are used for this purpose and that the threshold of safety varies with the type of infection, the distant symptoms, and the individual upon whom it is carried out.

Where distant reactions follow manipulation of the gland, the threshold of *toxin tolerance* becomes an extremely important matter. With lesions of the heart, the eye, and in the presence of an active arthritis, this threshold is very low and *one should treat the gland with the utmost gentleness* lest he do great harm. It is easy to prove that in these conditions one should so regulate the amount of pressure on the gland in his study and treatment that reactions are avoided, if he would help and not harm his patients. I have known a patient with focal infective iritis to be almost blinded by the reaction following a rough prostatic massage and patients with myocarditis almost to die, and I have seen patients with massive arthritis given a chill, a temperature of 104 or 105° F, and made seriously ill for days by a single firmly performed massage. In other words, one must think of prostatic manipulations in these cases as questions of toxin tolerance and dosage, and regulate pressure upon the gland so that the amount of toxin forced into the system is below the patient's reaction dose.

One should be extremely careful about such manipulations in the presence of prostatic hypertrophy of any great size. Not a few such patients have large varicosities at the vesical outlet, and it is then a simple matter to cause troublesome and even dangerous bleeding. A word of caution also should be given against massaging any prostate exhibiting firm nodulations. Most of these are carcinomatous and some few are tuberculous.

Prostatic massage should be carried out at three- or four day intervals and progress should be determined by microscopic study of the secretion. If there is no obvious reduction in the number of pus cells after six weeks of treatment, the patient again should be studied for further oral infection. No matter how much pus there may be in the prostatic secretion of one having tuberculosis elsewhere in the body, it is unwise to carry out such prostatic treatments. This procedure has been known to cause acute miliary tuberculosis and massive tuberculosis in other structures in tuberculous patients who gave no discernible evidence of tuberculous involvement of the prostate.

If, at the end of three or four months of treatment, the prostatic secretion still contains an excess of leukocytes, it is well to place the patient on a six to eight weeks' period of rest from treatments, during which time it is no waste of effort to have him carefully studied for other more distant infections which may be acting as retarding influences. Under the methods outlined it is possible to bring all but a comparatively small number of prostatic infections to the point of cure. As recurrences of oral infection may cause later reinfections of the gland, it is wise to restudy such patients at intervals for several years after the prostatic secretion has been rendered normal.

FIBROUS PROSTATIC BAR

The term "prostatic bar" has been so thoroughly confusing in the past that it probably is clearer to place the word "fibrous" before it to signify that one no longer is including what used to be called "glandular bar." The work of Randall very clearly demonstrated that this latter type of bar is, in reality, median prostatic lobe hypertrophy and bears no microscopic resemblance to the true fibrous bar here considered.

Age Incidence. For some years it has been taught that, in general, this lesion belongs to a much earlier period of adult life than does true prostatic hypertrophy. To some extent this is true, but the percentage of fibrous bars in Randall's series of autopsy cases, as compared with hypertrophy, fails to indicate that the bar belongs to early adult life.

Of the cases of bar occurred before that age as against 12.5 per cent of hypertrophy. Certainly no lesion that shows approximately a 69 per cent incidence beyond age fifty should be considered to belong to early adult life. This becomes even more evident if one considers that numbers of years do not always serve as safe indications of physiologic aging and that many men between forty and fifty are older than their numerical age suggests. Thus, if one makes his dividing line at forty years he finds that 87.5 per cent of fibrous bars occur after that age (see p. 87).

Etiology. Apparently the initial cause of median fibrotic bar is longstanding infection of the prostate wherein large numbers of round cells are deposited in the extrafollicular stroma. While this condition is in no sense confined to the portion of the prostate underlying the vesical outlet, it is this portion that causes all of the symptoms. The gradual transformation of these cellular deposits into fibrous tissue, with its inherent quality of later contraction, usually serves to reduce the bulk of the entire gland. In doing this it draws the posterior vesical "lip" upward as it narrows the lumen of the vesical outlet and thus causes a definite obstruction to the emptying of the bladder.

For many years it has been thought that gonorrhea accounted for most cases of fibrous bar formation. That such is the case is rather doubtful except, perhaps, where true abscesses of the gland have taken place. Since this is rare in the absence of direct trauma to the gland, it would be safer to say that in most cases it is the trauma that caused the abscess and that it is gonorrhea plus trauma that lays down the extrafollicular infiltration of round cells which eventually causes the bar. Furthermore, as most cases of chronic prostatitis are focal infective and not gonorrheal in origin and as both types, devoid of trauma, are intrafollicular inflammations, it is the trauma added to the inflammation rather than the inflammation alone which is etiologically responsible.

Pathology. The local pathologic process is that of interfollicular fibrosis wherein the stroma of the gland, although not actually increased in quantity, seems to make up a far larger part of the gland structure, owing to the contraction upon the follicles. Gradually as the obstruction at the vesical outlet and the difficulty in urination increase, the musculature of the bladder throws the mucosal wall into either fine or coarse trabeculae.² Between these hypertrophied muscle bands the mucosa may be pushed outward by the back-pressure of straining to empty the viscus so that shallow pockets called

cellules' or even true vesical diverticula develop. Where the backpressure has been such as to force the ureteral orifices, the ureters and kidney pelves may share in the dilatation and, in advanced cases, kidney function may be seriously affected.

Symptoms The symptoms of median bar formations are in practically all particulars the same as those of so-called hypertrophy of the prostate. As with this latter condition, they vary greatly in severity in different patients—some patients have urinary symptoms early and others have almost none until the obstruction is well advanced. The most common symptoms are difficulty in starting the urinary stream, the passage of a small stream lacking in trajectory, diurnal and nocturnal frequency, and a feeling as if the bladder had not emptied at urination. Complete retention is unusual, although it does occasionally take place. If infection of the urine occurs, pyuria, greater urinary frequency, vesical discomfort, and, at times, systemic evidences of toxemia are added to the picture.

Diagnosis The diagnosis is *solely cystoscopic* and rests upon the rather typical changes at the vesical outlet and in the posterior urethra. In typical cases there is a transverse crease in the trigone about midway between the vesical outlet and the interureteric line. The posterior vesical rim is pulled upward and there is a definite shortening of the postmontane fossa wherein the verumontanum is pulled much nearer to the median prostatic commissure than is normally the case. In marked cases, hypertrophy of the interureteric ridge, trabeculation of the bladder wall and, not uncommonly, considerable residual urine occur. Frequently the lateral prostatic lobes are pulled in so that they bulge prominently into the posterior urethra, giving a picture in miniature of that obtaining in lateral lobe hypertrophy.

In true bar formation this cystoscopic picture is a fixed one whether the patient strains or relaxes during the examination. The cystoscopist should be extremely careful in this regard, as it is possible for a straining patient to produce the typical changes of early median bar and to iron them out completely in a moment by thoroughly relaxing. This fact seems not to be generally known and, beyond the slightest doubt, many patients have received operations they did not need because of this muscular behavior and the fact that they did not happen completely to empty their bladders prior to the cystoscopic study.

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often set her stage to fool the unwary cystoscopist and lead the patient to an unneeded surgical procedure.

Treatment The treatment of fibrous median bar is removal of the obstructing tissue. It is here that the so-called electric excision finds its greatest usefulness. Carefully done, this operation carries a decidedly low mortality and morbidity rate. It rarely has to be repeated.

in the later stages of the disease, the patient may at times

vanced cases with any amount of residual urine. More often than not it eventually causes infection of the retained urine and adds greatly to the patient's discomfort and dangers.

Most careful urologists are agreed that the outstanding symptom calling

for operation is a steadily mounting residual urine. Some few insist upon operation as soon as the diagnosis is made. This latter view overlooks the fact that many patients with no residual urine and a typical vesical outlet picture of bar live comfortable lives with practically no urinary symptoms and never have a residual urine, and die of old age without having developed either.

PROSTATIC HYPERTROPHY

By far the most common cause of obstructive uropathy of middle life and beyond is what we have come to call prostatic hypertrophy. Although the term "hypertrophy" is perhaps not altogether an appropriate one,² its use has become such a fixed matter that there is little need for us to try to change it. The term, however, has been the cause of much confusion in the minds of physicians, for they have visualized an overgrowth of all of the glandular elements rather than the presence of a benign new growth which starts in one or several portions of the prostate and pushes the uninvolved true prostatic substance aside as it increases in size. Because these new growths may form in one or several portions of the gland, there occur quite an assortment of types or combinations thereof further to confuse the physician and, often, to render correct diagnosis impossible without the use of the cystoscope.

Etiology. Despite a wealth of study extending over many years, the true cause of prostatic hypertrophy still remains unknown. Many theories have been advanced to explain the lesion ranging all the way from infection to the reputed slowing down of male sex hormones (See p 251).

For quite a few years those who attributed the lesions to chronic infections of the prostate seemed to gain followers, despite the evidence contradicting such an etiology. The history files of any urologic office are filled with conclusive evidence that infection could *not* be the cause underlying the development of these benign growths which, during their formation, push surrounding tissues aside and from which they can be shelled by a definite line of cleavage as if they had a definite capsule. The mere fact that a large proportion of the glands showing such new growths are infected is certainly not suggestive of an infective etiology. As a matter of fact, about an equal number of infected prostates occur in persons who have no hypertrophy.

Further than this, almost every urologist of long experience not only has seen such new growths occur in glands that gave no evidence of ever having been infected, but also he has seen infected glands that became normal as the result of treatment, remained so for years, and then became hypertrophied, and probably no careful urologist has seen prostatic massage, either as a so called prophylactic measure or as a treatment for existing infection, have the slightest influence in preventing hypertrophy or in permanently changing it for the better once it has developed.

There seems to be more theoretical reason why hormone imbalances of one type or another might play a part in the etiology of this lesion. While some have reported the most remarkable results from hormone medication, others, using the same things, have been unable to duplicate them. There is need for extensive and carefully controlled study of the relation of the prostate to the endocrine system. At present, reported findings are so contradictory that they confuse rather than clarify the picture (See p 251).

Pathology. Aside from the formation of benign new growths in the gland substance, the majority of which start in the suburethral tissues either of the lateral or subcervical lobes, most of the important pathologic changes of prostatic hypertrophy are found in the bladder and upper urinary tract. The bladder wall attempts to overcome the obstruction to its proper emptying, hypertrophy of its muscular coat usually supervenes, so that the fasciculi stand out prominently, and between these enlarged bands the bladder wall is pushed out to form cellulæ or, even, true diverticula. As the obstruction increases the amount of residual urine increases, and unless the obstructing tissue is removed surgically by one means or another, the kidneys are forced to work against increasing amounts of positive pressure. Thus, renal function gradually becomes impaired, waste products are retained in the blood stream and true uremia sets in. (See p. 595.)

Symptoms. It is unfortunate that some of the symptoms of prostatism also are present in countless individuals who have not the slightest structural interference to proper emptying of the bladder. This is particularly so of diurnal frequency of urination, the need of emptying the bladder during the night, slowness in starting the urinary stream, and even marked reduction in the size and force of the stream. Indeed, it is not uncommon to encounter all of these symptoms in persons who have no other cause for them than psychic meddling with the delicately balanced mechanism of urination. This is particularly true of many of the aged because prostatic hypertrophy has attracted so much attention among the laity.

Functional changes are so common in the years beyond the mid forties that they well might be called *normal*. When these attract the attention of the self-analyst they may give him quite an assortment of fears to convince him that all is not as it should be. Just as soon as these enter the mind they may become fixations that are none too good for the patient. Almost before the sun sets he has become urination conscious and a symptom complex may entrain that, without careful study of the vesical outlet, could convince almost anyone that some operative procedure was demanded. I regret that the limitations of this chapter do not permit so full a discussion of these urinary vagaries as appears in the chapter on the psychic factors of urogenital symptoms in *Office Urology*.³ That the mind very commonly does cause such symptoms seems not to be generally realized. The lack of this knowledge has caused many a faulty diagnosis, as well as added greatly to the discomfort and anxiety of many whom psychotherapy alone would have cured. Indeed, from the standpoint of symptoms alone this psychogenic group is probably larger than is that wherein true prostatic pathology is the underlying cause.⁴

This group having what rather appropriately might be called "psychic prostatism" is important and large. Hence the physician should not only understand its underlying causes but also realize that its members cannot possibly be separated from many of those who have a true prostatic hypertrophy unless the most careful studies are made, studies that should include a cystoscopic examination. For one to assume that he safely can tell the real from the unreal in this regard by symptoms alone is to attribute to himself a degree of diagnostic power that has long since been abandoned by every urologist of experience and is quickly deserted by the merest novice in things urologic.

Turning, then, to the symptoms of the group with *true hypertrophy* we

find that, even in the presence of marked prostatic enlargement, some patients have no symptoms to call attention to the fact, while others who have only minor grades of enlargement have highly annoying symptoms. In the main, these differences usually rest in the location rather than the size of the new growth. It is outstanding that new growths which involve the median lobe commonly are the ones causing the most pronounced symptoms because they are more highly obstructive and, as a result, are usually associated with varying amounts of residual urine. On the other hand, it is not uncommon that when the new growths involve only the lateral prostatic lobes the prostate may become enormously enlarged without inducing enough urinary symptoms to arouse suspicion until, perhaps, an attack of acute retention of urine dramatically enters the picture. Thus, there is a wide variation in symptoms and their severity, and the patient with the largest growth may have the mildest of symptoms or none at all—a fact that gives great value to prostatic palpation as a routine procedure during middle and later life. Not that such palpation, as will be pointed out in the section on diagnosis, is always diagnostic. More commonly it is found that where such palpation holds the least diagnostic value the patient has the more pronounced urinary symptoms and these, in themselves, urge more extensive study.

The most common early symptom of prostatic hypertrophy is the necessity to empty the bladder one or more times during the night. Though it is so common as to be almost the rule that men beyond middle life empty the bladder once during the night, most of these have little difficulty in starting the stream promptly. The prostatic, so-called, usually has considerable difficulty getting things started and, when the stream does flow, it is seldom a very vigorous one. The act commonly is completed in spurts and often followed by considerable dribbling.

If there is considerable residual urine pain of distention usually is present and the patient feels that he does not completely empty his bladder. As this residual increases, frequent urinations during the day become necessary and the more the patient concentrates upon the matter the greater becomes his frequency of urination. Often he develops the habit of straining to start the stream, although in some cases an urgency occurs that overcomes this need and makes distance to a urinal one of his greatest hazards.

Attacks of acute retention, particularly in lateral lobe hypertrophy, may follow exposure to cold, indulgence in alcoholic beverages, failure to empty the bladder for too long a period of time or anxiety.

Bleeding from mucosal varicosities at the vesical outlet is not an uncommon symptom. This may cause a definite hematuria or may be confined to a few drops of blood as the urinary act is completed.

Perhaps no one has more tersely described these varied groups of symptoms than has Keyes⁵ and his words are well worth verbatim quotation:

"The Usual Type" The symptoms begin with nocturnal frequency and difficulty in urination. This increases gradually and the patient passes on through the first, second and third stages as described below.

"The Acute Complete Retention Type" Acute complete retention of urine may supervene at any moment in the course of the disease. It is the first symptom of importance in almost half of the cases.

"If properly treated it may sometimes be relieved, and the bladder resume its ability to empty itself satisfactorily. But if the infection cannot be

controlled or if some residual urine remains the acute complete retention will soon recur. Very few patients with acute complete retention escape further trouble within a year. Yet they may escape for a number of years without any further symptoms.

Urinary Toxemia The patient with a tolerant uninfected bladder may have an acute complete retention of urine and may not concern himself about his gradually increasing frequency of urination. He may thus reach a condition of chronic distention of the bladder and kidneys without consulting a physician. Under such circumstances the first condition that he notes is a loss of weight and strength accompanied by constipation and dry mouth. Even these he may not note until slight infection adds fever to his symptoms. Such patients unless carefully examined may be treated for digestive disturbances for a considerable time though the mere laying of a hand upon the lower belly identifies the distended bladder and intelligent investigation of the patient's history reveals polyuria, frequency of urination and dry mouth.

Diagnosis As can be understood readily from the foregoing the diagnosis of prostatic hypertrophy is not always the simple matter that it rather generally seems to be considered. At least one's clinical experiences would lead him to the conclusion that medical minds have not always sensed the complete diagnostic picture and unquestionably they have attributed to digital palpation alone a value far in excess of its possibilities. *Palpation* alone is of positive value in only about three fifths of the cases of vesical outlet obstruction other than abscess and carcinoma. Of 279 cases of such obstructions 27.4 per cent were due to median lobe obstruction and 13 per cent to subcervical gland hypertrophy.¹ These two types of hypertrophy extend toward the urethra or bladder and give rectal palpation alone an approximate 40 per cent margin of error for they present no rectal evidence of their presence.

Thus it is evident that in a large percentage of cases diagnosis must rest upon *cystoscopic study* and even where rectal palpation does demonstrate hypertrophy such study is the only means whereby a true and complete evaluation of existing conditions can be made. Of course one should not urge such a study for every case of prostatic hypertrophy any more than he should say that no patient with hypertrophy should be subjected to cystoscopy as some do. Cystoscopy like all instrumental procedures has its indications and its contraindications. It is because these latter have not always been followed that some say sweepingly that cystoscopy should not be carried out in any case. Such an extreme stand is certainly not in step with modern views. Today it is safe teaching to say that barring definite contraindications every patient who has or is suspected of having a vesical outlet obstruction should have a cystoscopic study carried out with all of the safety precautions that modern knowledge has lent to the art.

It should be borne in mind constantly that unless strict asepsis is employed the passage of a catheter into a bladder containing residual urine is extremely hazardous. Indeed it is not altogether without danger no matter how careful the asepsis. The passage of a cystoscope in gentle hands holds no more dangerous possibilities than simple catheterization and it gives a
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Thus, the diagnosis of prostatic hypertrophy rests upon the symptoms and rectal palpation, and cystoscopic study wherever it can be carried out without great danger to the patient. In an evaluation of the patient's symptoms it is unwise to place too much importance upon the fact that many of them can be psychogenic until other possible causes have been definitely ruled out by a careful and complete study. This, alone, would prevent much morbidity and save not a few lives.

Treatment. Although hormone studies are revealing much of interest regarding prostatic hypertrophy, it cannot be said that we have reached a point where such therapy has outmoded operative procedures. About as much as science has been able to accomplish to date is a reduction of symptoms in a few cases but not a reduction of the hypertrophy.

As far as true prostatic hypertrophy is concerned, it would be ideal if every such new growth could be surgically removed by one type of operation or another. Just what operation is advisable depends upon a number of things, not the least of which is the skill and judgment of him who carries out the particular procedure. The practitioner would do well to realize that operations were devised for patients and not patients for operations. This is of particular importance in these days wherein patients so commonly insist upon a particular type of operation which may not in any sense be the one needed.

The fact that most of the present morbidity and mortality due to benign hypertrophy occurs in those patients who should have been operated upon years earlier, emphasizes the need for greater concern and less procrastination on the part of the physician. Prostatic operations are not done just because the gland is enlarged, but because of the great damage such enlargements may do to the bladder, the upper urinary tract, and the general system. It is a mistake to delay beyond the time when this damage first becomes evident.

It is, of course, realized that there are some patients who, when first seen by the physician, are no longer fit subjects for any type of operation, although modern urologic procedure, better preoperative preparation, and improved geriatric medicine have reduced their number greatly. Even in those of advanced years, operation has been and can be made a far safer matter than was the case but a decade ago. Thus, age and present physical condition should not always loom too greatly as contraindications to operative procedures for the relief of such obstructions.

For the practitioner to use these arguments as excuses to place patients upon *catheter life* is a grave injustice to the patient. Such questions should be answered by those whose special training fit them to do so, and, if catheter life is all that can be held out for the given patient, the physician should see that it is carried out with all the safeguards now possible. Whether intermittent or indwelling catheterization is advisable must depend upon a number of things, not the least of which is the temperament of the patient himself. The menace of placing the catheter in the hands of the patient is great and, wherever possible, the physician should do the catheterizations. The technic, precautions, and dangers of catheter life can be found in any standard book upon urology and should be thoroughly mastered by him who essays to guide such patients through their remaining days which, as a rule, are neither many nor happy ones.

PROSTATIC CARCINOMA

Carcinoma of the prostate gland rarely makes its appearance before the age of forty-five years. Of all urologic lesions it is, unquestionably, the most deadly. Seldom, indeed, is cure accomplished and then only because radical surgery could be done in those extremely rare cases in which a diagnosis had been made before metastasis has taken place. More usually it mocks the urologic surgeon, the radiologist, and all of the curative agents that science so far has devised.

It is the rather general medical impression that these patients are all doomed to lives of pain and urinary dysfunction of the most devastating character. That such is the case with some patients is not to be denied, but that severe and continuous suffering is the lot of all is far from the truth, except perhaps in the terminal few weeks. The striking results with estrogenic hormone (diethylstilbestrol) therapy first reported by Huggins⁶ have been amply and repeatedly confirmed.⁷ Estrogen therapy, either alone or with concomitant castration, now offers from six months to two years greater life expectancy in prostatic carcinoma, even when multiple metastases exist.⁸

It has been shown that the prostatic epithelium contains many times more acid phosphatase than does any other tissue of the body. The normal blood serum content of this substance is enormously increased in the presence of bony metastases from prostatic carcinoma. When, under such circumstances, testicular function is depressed by the use of estrogenic hormones (stilbestrol) the blood concentration of acid phosphatase drops markedly.⁶ When, however, both testicles are removed surgically it falls to normal and remains there.

While this does not in any sense answer the question of etiology, it does suggest that further study may lead us much closer to the facts.

Pathology. Carcinoma may develop in any part of the prostate gland, although it is extremely rare in the anterior lobe. It occurs most often in one or both lateral lobes. The next most common site is in the posterior lobe, far less commonly does it start in the subcervical lobe. Beginning as a small area, it gradually enlarges and spreads into other portions of the gland. At first a definite nodular limitation of the growth may occur. Gradually this enlarges to fill the entire capsule and, in some cases, the structure assumes a size sufficient to extend from one lateral wall of the pelvis to the other and so far up under the base of the bladder that the intrarectal palpating finger cannot reach its upper limit. The growth is of almost stony hardness which, aside from size in some cases, is its most outstanding diagnostic feature. In many cases the growth causes a true obstructive uropathy because it develops at the vesical outlet. Vesical involvement is not altogether uncommon.

Metastasis occurs early, being present in all but a few cases before the diagnosis is made. Most of the early metastases are confined to the bony system, particularly the pelvis and the bodies of the vertebrae. The lungs, pleura, pancreas, retroperitoneal lymph nodes, and other structures not uncommonly are involved.

Hammond divides prostatic carcinomata into three types as follows:

"(a) The *acute fulminating type*, that resembles an acute inflammation in its onset and spreads, and leads to death in a few months.

"(b) The *disseminating type*, where though the growth is small, general metastases are present almost from the start.

'(c) The *scirrhus type*, where the growth remains localized for a long period and dissemination to the glands and to the viscera occurs later. This latter type is by far the most common."

Clinical Course. Because of these variations in type, it is natural that there should be many variations in clinical course, and, owing to the dramatic character of the less usual types, it has become a rather common custom to pose them as the standards by which the usual clinical courses are judged. In doing so, we have lost sight of the fact that, in the vast majority of the cases, the growth has been present for months or even years before the diagnosis has been made—periods in which the patient seemed in health and was in comfort as far as subjective symptoms were concerned. Indeed, the condition rarely is suspected until symptoms occur due to vesical outlet obstruction or to metastases.

With the *scirrhus type*, patients frequently lead comfortable lives for some years before cachexia sets in. I can recall patients who kept in such health and comfort for from three to five years after the diagnosis had been made that the patient's family and physician did not hesitate to condemn the diagnosis. Upon several occasions I almost began to doubt its correctness myself as, by streak of human perversity, the victims seemed commonly to cross my path. Each, however, eventually proved the correctness of the diagnosis by going the way of all such patients. Some of these were cases of the *scirrhus type* with pelvic cavities rather well filled with growth at the time of study. Others were discovered in the earlier stages and their conditions only admitted by others when the growth was of enormous size.

Symptoms. Prostatic carcinoma is essentially a *silent, painless lesion* until the growth either obstructs the vesical outlet, invades the bladder, or metastasizes to distant structures. Thus the symptoms of the lesions are those of *cystitis, obstructive uropathy*, or the *referred nerve pains* of metastatic lesions. Almost never is there pain in the gland itself.

Diagnosis. The diagnosis of prostatic carcinoma most commonly is made in the course of studies to explain the symptoms due to obstructive uropathies or metastasis. Not infrequently the discovery of bony involvement at x-ray is the first significantly suggestive finding. A few cases are discovered through routine diagnostic study of the prostate.

The outstanding diagnostic feature is the stony hardness of a part or all of the gland. In most cases the carcinomatous prostate gland is so fixed to the pelvic walls when discovered that it has no mobility whatever to the examining finger.

In the acute fulminating type this stony hardness is not so striking and may, indeed, be absent. In the other two types it always is present when the growth has reached the fibrous capsule. One should not, however, lose sight of the fact that some normal prostates are of such form and consistency as to raise a question of malignancy. Also, it should be borne in mind that the markedly tuberculous gland may be of stony hardness. In this latter case, however, there are other evidences of urogenital tuberculosis to settle the diagnosis.

Wherever the least suspicion is aroused, it is wise to resort to roentgenography for search of bony metastasis. If these studies are suspicious but not positive the presence of an enormously high blood serum acid phosphatase content should settle the doubt.

Treatment So far as the question of cure is concerned the outlook is, at best, dismal. Total prostatectomy, before metastasis has occurred, seems to have been curative in the hands of some urologic surgeons. Unfortunately, only a few cases are discovered early enough to fulfill this qualification.

Beyond this stage surgery to the gland itself has nothing to offer from a curative standpoint and he is a wise physician who does not allow family or patient anxiety to crowd him into advising it. For this is one disease in which the drama of the operating room more often than not adds to the tragedy of the patient's remaining days. About the only call for direct surgery to the gland in these cases is for the relief of vesical outlet obstruction and for this purpose transurethral electric excision is the procedure of choice. Patients tolerate it well, as a rule, and commonly obtain great urinary relief from it. In not a few cases it must be repeated from time to time.

Irradiation with either radium or x-ray has no curative influence. The latter, at times, is of service to relieve the pains of metastasis and to check the bleeding from the vesical involvement.

The most promising procedure offering relief of symptoms in such cases is a *bilateral orchidectomy*. Though the disease is not cured, the patient is quickly relieved of his pain, improved physically, and leads a far more comfortable life thereafter. Certainly this is a great gain for many of these unfortunates and it is to be hoped that further experience with it upholds the present promise.

Diethylstilbestrol orally may yield striking results in recession of pain, increased vigor, and actual diminution in the size of roentgenologically demonstrable bony metastases. A few such patients, perhaps receiving unnecessarily large doses, develop gynecomastia. Where concealment of the diagnosis from the patient is desirable, estrogen therapy can be administered with evasive explanations such as "for arthritis"; it is difficult to explain the reasons for orchidectomy without revealing the true diagnosis. Often it is a delicate question of balancing morale against slightly better somatic results.

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CHAPTER 41

GERIATRIC GYNECOLOGY

E C HAMBLÉN

THE climacteric, or gynecologic aging, is unique because of its specific hormonal etiology—namely, hypoestrogenism, which results from a gradual and irreversible aging of the ovaries

In this presentation, the following general phases of geriatric gynecology are discussed

- (1) The overall clinical characteristics of the climacteric
- (2) The initial, compensatory, and remote alterations of the endocrine system
- (3) The effects of progressive hypoestrogenism on the genital organs
- (4) The effects of climacteric hypoestrogenism on non-genital organs and systems
- (5) The effects of this hypoestrogenism on preexisting constitutional disease and local pathology
- (6) The predisposing effects of climacteric hypoestrogenism to constitutional disease and local pathology
- (7) The clinical course of the climacteric
- (8) Hygiene and therapy

OVERALL CLINICAL CHARACTERISTICS OF THE CLIMACTERIC

The reproductive epoch lasts, as a rule, some twenty-five to thirty-five years. It is terminated by a phase of sexual regression, which lasts a varying number of years. This sexual aging causes a loss of the childbearing function, a cessation of menstruation, and involutional changes in the genital system. Libido and coital function may be undisturbed for a number of years after the onset of the other evidences of sexual aging. Physical well-being should continue for years unless it is impaired by somatic senescence.

Duration. The term, *climacteric*, is preferred to the term, *menopause*, as it symbolizes the gradual nature of the aging process. Menopause, the cessation of bleeding, is only one isolated event in sexual regression. The climacteric begins with the first break in normal ovarian function and it lasts until the endocrine and gynecologic adjustments to ovarian senility are completed. Accordingly, the climacteric begins in advance of the menopause and it lasts long after the menopause. For convenience, two stages of the climacteric may be defined—the *premenopausal* and the *postmenopausal* stages.

Time of Menopause. Uterine bleeding usually ceases between the forty-fifth and fiftieth years. The menopause of most women in this country occurs between the forty-seventh and forty-eighth years. Some clinicians believe that menopause now occurs earlier than formerly.

Factors which influence the time of menopause include the following: racial peculiarities, heredity, general health, social status, climate, parity, and

gynecologic disease. It is said that business and professional women have early menopause.

Premature Menopause Some of the common causes of premature menopause include the following: surgical castration, irradiation of the ovaries, prolonged lactation, debilitating diseases, and various endocrinopathies which result in hypoestrogenism. Although hysterectomy brings about cessation of menstruation, it is not followed promptly by any evidences of ovarian deficiency, if the ovaries have been conserved.

The differentiation between intercurrent amenorrhea during the reproductive epoch and premature menopause is not easy. The estimation of the daily urinary output of gonadotropins is very helpful. When the urinary gonadotropins are found markedly elevated (increased by five fold or more) a diagnosis of premature menopause is warranted. Hypergonadotropinemia of this order occurs only when there is marked intrinsic failure of the ovaries.

Delayed Menopause It is said that menopause is delayed past the fiftieth year in 10 to 12.5 per cent of women. Any postponement of menopause past the age of fifty-five years, however, should lead to a thorough gynecologic examination. Bleeding of genital malignancy may pass for a delay in menopause. Undiagnosed granulosa cell tumors of the ovary may explain some instances of unusual delay of menopause.

Induced Menopause There is no universal accord concerning the symptomatology of spontaneous climacteric and of induced menopause. Many women experience few subjective symptoms following prematurely induced menopause. In general, it is believed that artificial menopause, being abrupt in its onset, may provoke more severe symptoms but that these symptoms are shorter in duration.

ALTERATIONS OF THE ENDOCRINE SYSTEM

Specific functional and structural changes in the ovaries are the initial and most striking of these alterations. Sequential to an increasing hyporeactivity of the ovaries, progressive and compensatory changes occur in the anterior lobe of the pituitary gland. In addition, there are associated and significant alterations in the thyroid, adrenal cortex, and adrenal medulla.

Ovaries Both the germinal and the endocrine functions of the ovaries fail during the climacteric.

Many years ago Waldeyer showed that ova ceased to be present in ovaries near the time of menopause and thereafter. Recent studies^{1, 2} have confirmed these observations: no ova were found in sections of ovaries of women at or near the menopause, and furthermore, ova are scarce during the latter third or fourth of the reproductive epoch. Accordingly, if the concept is accepted that oogenesis occurs from germinal epithelium, then this process fails as menopause approaches.

The endocrine failure of the ovaries is gradual and progressive. It results from a growing refractivity of the ovaries to the gonadotropic stimuli of the anterior lobe of the pituitary gland.

At first, this refractivity is moderate and relative and it does not involve all of the various gonadotropic stimuli. The sequence of events doubtlessly approximates the following. The initial break probably involves the corpus luteum phase of the cycle. Proof is not clear cut, it seems reasonable how

ever to envision variations in the length and intensity of corpus luteum action which probably result from refractivity to the luteotropic action of the pituitary and perhaps are associated with a defective unfolding of the corpus luteum. In view of the germinal failure of the ovary previously discussed anovular follicles may erupt and give rise to defective corpora lutea which may result in hypoprogesterinism and in short luteal phases of cycles with bleeding from insufficiently or incompletely prepared progestational endometria. Studies of endometrial biopsies and of basal temperature curves should provide interesting data. It is doubted however that these variations in progesterin secretion produce any significant alterations in the cycle or in the duration and character of bleeding.

The next break however is very important. It is characterized by a failure of ovulation. As a result the progestational phase of the cycle no longer occurs and corpora lutea are absent. The ovarian cycle accordingly becomes uniphasic and is characterized solely by estrogen secretion incidental to the growth and regression of follicles. Thus the climacteric woman becomes sterile not only because of failure of oogenesis but also because of failure of ovulation. Woman loses therefore her reproductive function before there is any significant decrease in estrogen levels. Anovulatory bleeding of the premenopausal phase of the climacteric differs from menstrual bleeding in several respects. First it occurs from interval or estrogenic

is a stabilizing influence in menstrual chronology. The cycles which are monitored solely by the waxing and waning of estrogen levels resulting from follicular maturation and regression are characterized by various irregularities. The amount and duration of bleeding however may not be significantly altered. During the early stages of this anovulatory failure degrees of reversibility may be encountered. Moderate degrees of refractivity may be overcome by early and moderate compensatory hyperactivity of the pituitary gland. The result may be that occasionally at the termination of follicle maturation an effective amount of luteinizing hormone may be released from the pituitary with resultant ovulation and corpus luteum unfolding. These isolated recoveries of function may explain unexpected pregnancies during immediately premenopausal years.

The third and final break in ovarian function is characterized by estrogenic failure. For a variable length of time a number of months or even sever-

early how-
enough granulosa proliferation and follicle maturation to maintain effective estrogen levels. Accordingly the gonadotropic function of the pituitary constantly increases in an effort to overcome this refractivity. Eventually waves of estrogen secretion have progressively decreased amplitudes. The phase of

basal secretion of estrogen may continue for a number of years after the menopause

Once the course of ovarian senility is well under way, it is irreversible. The gonadotropic activity of the pituitary of a woman in the climacteric may be from five- to fifty-fold that of a woman during the reproductive epoch. If this degree of pituitary stimulation cannot reverse the ovarian aging, meager attempts at gonadotropic therapy are quite futile. Indeed, reactivation of aging ovaries involves not only a rejuvenation of estrogen producing cells (granulosa cells in particular) but also of the ova-producing elements (the germinal epithelium).

These functional changes have their anatomic components. As the menopause approaches, the ovaries contain decreasing numbers of ova. Eventually follicles are present which contain no ova. During the phase of hypoprogesterinism, irregularities of corpus luteum proliferation may be evidenced. During the phase of anovulatory failure, no corpora lutea may be observed or only an occasional one due to a temporary recovery of function. Follicular cysts in various stages of regression remain in lieu of corpora lutea. These cysts contain no ova. During the phase of estrogenic failure, progressively less evidence of follicular maturation is encountered. The ovaries shrink in size and the follicles are small. Eventually the ovaries become quite small and sclerotic. Finally they constitute little more than folds of tissue and are devoid of any evidences of function.

Pituitary The pituitary is unique during the process of aging. When other organs are regressing and their functions are being impaired by senescence, the pituitary characteristically becomes hyperactive. Data in this regard are conclusive and clear cut. Implantational studies with human pituitaries indicate that contents of follicle stimulating and luteinizing gonadotropins are definitely increased. Urinary hormone estimations likewise reveal a marked increase in the excretion of follicle stimulating gonadotropin and a very significant increase in that of luteinizing gonadotropin. Data on luteotropic gonadotropin have not been reported. (See p. 249.)

Anatomic and cytologic studies bear testimony to this hyperfunction of the pituitary during climacteric. It is reported generally that the gland is enlarged during and after the climacteric. Severinghaus³ has made detailed cytologic studies of the pituitary of climacteric and aging women, taking care that the cellular components were studied from the standpoint of secre-

increasing hypofunction, but instead a gland which gives evidence of secretory activity even greater than in the period of maturity preceding the menopause. In this activity both acidophiles and basophiles are involved and we must ascribe to the cells a high rate of elaboration as well as a release of their secretory products. This reversal in the usual trend of changes which comes with increasing age has not been found in the hypophyses of men or of those in either sex of other species which have been studied."

It is unlikely that the hyperactivity of the pituitary during and after the climacteric is confined solely to gonadotropic activity. An increased urinary excretion of pituitary thyrotropin has been described. In addition, experimental studies have shown that oophorectomy may be followed by an

increased secretion of thyrotropin, by an elevation of blood iodine values, and by associated alterations in basal metabolism. Clinically, menopausal or climacteric hyperthyroidism is not uncommon.

Objective data on the level of other pituitary factors during the climacteric are not available. Recent studies by Reifstein and his associates⁴ indicate that luteinizing gonadotropin has a corticotropic action which involves the secretion of the so-called "N hormones" of the adrenal cortex. These so-called "N hormones" embrace the sex and other steroids which stimulate nitrogen metabolism. Accordingly, the increased output of luteinizing hormones during and after the climacteric may result in a hypersecretion of so-called "N hormones" by the adrenal cortex. This increased secretion should result in increased urinary excretion of 17-ketosteroids. No data have been supplied that true adrenocorticotrophic hormone values are increased at this time. The adrenocorticotrophic hormone has been related to secretion of the so-called "S hormones" of the adrenal cortex, which are concerned with

gonadectomy. Huggins,⁵ in studies of males who were gonadectomized because of prostatic carcinoma, found that subsequent compensatory hypergonadotropic action of the pituitary caused an increased secretion of sex steroids and that the outpouring of

so marked as to undo the beneficial effect of the castration. The hypergonadotropic action of the pituitary was related to the increased secretion of luteinizing gonadotropin by the pituitary.

The hypergonadotropic activity of the pituitary is compensatory and seeks to forestall ovarian senility so long as this is possible and, failing in this, to glean what sex steroids the adrenal cortex may be stimulated to secrete. It continues throughout the female climacteric and during somatic senescence, having been found to be increased in the aged.

that the adrenals are enlarged in the aged and that this enlargement follows gonadal regression.

Studies of urinary androgen levels, following oophorectomy, provide some information on adrenal function. Callow⁷ reported data, an analysis of which indicated that oophorectomy was not followed by any significant decreases in androgen secretion and that, indeed, androgen values may be increased.

Our group⁸ has reported that there occurs a transient increase in the total urinary 17-ketosteroids (which measure adrenal function in the female) during the climacteric and following bilateral oophorectomy. Huggins' studies, to which reference was made previously, confirm these observations. These increased 17-ketosteroid levels are due not to an increased secretion of follicle-stimulating gonadotropin by the pituitary but to an increased secretion of luteinizing gonadotropin. In pubescents of both sexes with hypergonadotropinemia, 17-ketosteroid levels are not usually elevated because the hypergonadotropinemia of these patients involves only the follicle stimulating hormone.

The adrenal cortex doubtlessly secretes small amounts of estrogens during

the reproductive epoch, however, at this time it is difficult to differentiate ovarian and adrenal estrogens. Climacteric and postclimacteric women continue to excrete small amounts of estrogen in the urine. This may be related to a basal secretion of estrogen by the senile ovaries. Oophorectomy, however, does not terminate this estrogen secretion.

Although corpus luteum activity ceases prior to the menopause and little progesterin, if any, is secreted by the ovaries after menopause, small amounts of pregnanediol are found in urine years after the menopause and after oophorectomy. This pregnanediol (a metabolic product of progesterin) doubtlessly is of adrenal origin.

Because of the evidence of climacteric alterations in the secretion of androgens and 17-ketosteroids by the adrenals and because of continued secretion of estrogen and of progesterin by the cortex during the aging process, our group has referred to the adrenals as the "gonads of the aged." We have suggested, therefore, that a compensatory increase in the cortical secretion of sex steroids occurs during the climacteric.

A medullary hyperfunction also may occur during the climacteric. Actual proof of this is difficult. It is known that many of the climacteric symptoms simulate those produced by an injection of epinephrine. Climacteric women have an increased sensitivity to epinephrine. As they become better adjusted to the climacteric, this sensitivity decreases. Since no medullotropic action of the pituitary has been established, a medullary hyperactivity cannot be related directly to pituitary hyperactivity. It may be related, however, to the increased hyperactivity of the cortex, since hypercorticalism may

Thyroid Glands. The thyroid gland is enlarged in the climacteric. This thyroid enlargement has been related by clinical and experimental data to hyperactivity. Blood iodine values are said to increase during the climacteric. Salter⁹ has related these increased blood iodine values to an increased outpouring of the thyrotropic hormone. Bilateral oophorectomy, however, has not been shown to produce any consistent elevations in basal metabolism.

Other Glands. Climacteric alterations in other glands apparently are not striking. The peaking of incidence of diabetes mellitus during the climacteric may suggest that pancreatropic activities may be involved in the hyperactivity of the pituitary.

Summary of Hormonology. The established, as well as the hypothesized, hormonal changes of the climacteric, are synthesized into the following step-by-step clinical narrative.

(1) **Phase of ovarian failure.** When the life span of the ovaries has been run, there develops an increasing and progressive refractivity to the gonadotropic stimuli of the pituitary gland. Ovulation and corpus luteum formation at first fail to occur. Accordingly, the cyclic influence of progesterin is lost and the pituitary no longer is modified by it. The ovaries continue to respond to the follicle stimulating gonadotropin with the result that estrogenic bleeding of regular or irregular nature follows when critical declines occur in the waves of estrogen secretion. Eventually the ovaries become so refractive to gonadotropic stimuli that there is little cyclic alteration in estrogen levels and uterine bleeding ceases.

(2) **Phase of pituitary hyperactivity.** The function of the pituitary, being

no longer conditioned by the cyclic influences of progesterin and of estrogen, is altered both qualitatively and quantitatively. It secretes progressively larger amounts of follicle-stimulating and luteinizing gonadotropin. As the estrogen waves of secretion decrease in amplitude, the pituitary gradually loses its cyclicity and functions at a continuously high level. Not only does this hyperactivity involve the gonadotropic functions of the pituitary but also the thyrotropic functions and, perhaps, other functions.

(3) Phase of thyroid and medullary hyperactivity. Unlike the failing and refractive ovaries, the adrenals and the thyroid are not unresponsive to the increased pituitary stimuli which reach them, and secondary hyperactivities of these glands are elicited according to diverse factors, which include local glandular receptivities and psychosomatic patterns. Both the adrenal cortex and the medulla doubtless share in this hyperactivity. Accordingly, two clinical possibilities exist for the causation of subjective symptomatology—this may result from hyperthyroidism or from hypermedullarism.

(4) Phase of cortical stabilization. Ultimate stabilization, and it is well known that the climacteric is a self-limited condition, has been related theoretically to an increase in cortical function, incident to an increased secretion of luteinizing gonadotropin by the pituitary. The subsequent increased outpouring of 17-ketosteroids, androgens, and, perhaps, estrogens and progesterin by the cortex may permit the exertion of conditioning influences upon the pituitary similar to those formerly exerted by the ovaries. Accordingly, pituitary hyperactivity may be curbed and the endocrine system stabilized, although at a higher level of function than formerly. This stabilization may terminate the symptomatic phase of the climacteric by curtailing further hyperactivity of the thyroid and adrenal medulla.

According to this endocrine schema, the sexual regressions which characterize the latter stages of the climacteric may be regarded as the result of both an estrogen deficiency and of a relative and absolute increase in androgens.

CHANGES IN THE GENITAL SYSTEM

The genital organs do not regress simultaneously with the menopause. Indeed, these regressions do not begin until a critical depression of estrogen levels ensues. Although the cyclic waves of estrogen secretion may be inadequate to provide significant endometrial growth and subsequent withdrawal bleeding, the basal secretion of estrogen may be quite ample to forestall genital regressions. There are individual differences among women. Some women may show marked genital regressions a few months following the menopause. Other women, on the other hand, may have little regression several years following menopause.

Ovaries. The ovarian changes have been discussed.

Fallopian Tubes. There is a general atrophy. The tubal walls are thin and the lumina are constricted. The epithelium becomes flattened and the plications and the cilia disappear.

Ligaments. The ligaments of the uterus involute, losing much of their muscular constitution and strength, and become thin fibrous bands.

Uterus. As hypoestrogenism increases, the uterus decreases in volume due to thinning and atrophy of its walls, because of the transformation of the myometrium, for the most part, into fibrous tissue. Accordingly, the uterus becomes small, firmer, and more fibrous. The size of the uterine cavity is

decreased. When myomata and fibromyomata are present, these share in the volumetric reduction of the uterus. Likewise the endometrium becomes extremely atrophic and thin. These uterine changes as a rule begin some time after the menopause and continue gradually for a number of years. Ordinarily, uterine regression begins before there is any significant vaginal atrophy.

Endometrial atrophy occurs before there is significant volumetric reduction of the uterus. During the premenopausal stage of the climacteric, there may be evidence of deficient estrogen stimulation of the endometrium. After the menopause, the endometrium shows various stages of progressive hypoeestrogenism. Eventually the endometrium is comprised only of a surface epithelium and a very thin dense stroma. This dense stroma may contain occasional small tubular glands. These small glands are lined by low epithelial cells. There are few vascular elements in the atrophic endometrium. Areas of endometrial denudation may be encountered and these may be sites of endometrial adhesions. At times these apposition adhesions may obliterate almost completely the uterine cavity.

Although the thin atrophic endometrium is the most common postmenopausal finding, there are distinct variations in the endometrial pattern after menopause. In some women the mucosa may be of varied thickness and not always very thin. In local areas, there may be found cystic and dilated glands, as described for so called Swiss cheese hyperplasia. Occasionally there may be an active hyperplasia, which may be diffuse or patchy. Novak¹⁰ has called attention to the possible relationship of this patchy hyperplasia to carcinoma of the endometrium. This local hyperplasia may occur in postmenopausal women who have no gynecologic disease. It has been suggested that it might be related to undue sensitivity of the aging endometrium to minimal basal supplies of estrogens which are still available either from the aging ovaries or from the adrenal cortex.

Cervix. The cervix undergoes volumetric reduction along with the uterine corpus. It becomes more fibrous, of firmer consistency, and often appears more pointed. As a rule the ultimate volumetric loss of the cervix is relatively less than that of the uterus, with the result that the cervicouterine ratio reverts to that characteristic of adolescence. The caliber of the endocervical canal is reduced and adhesions of the cervical mucosa may occur and further reduce the canal with resultant pyometra. Atrophy of the cervical mucosa parallels that of the endometrium. The glands lose their cilia, become more flattened and there occurs diminution in secretory function. Ducts of the cervical glands may become stenosed and retention cysts may develop. The mucous membranes covering the vaginal cervix appear smooth, white and glistening.

Vagina. Volumetric reduction of the vaginal cavity usually begins several years after the menopause. This reduction is particularly marked at the introitus. The cavity becomes tubular, due to the obliteration of the fornices and due to an increasing inelasticity of the vaginal walls. The rugae of the vaginal walls disappear and the mucosa has a dry and glistening appearance. There is a scanty vaginal fluid and fine vaginal adhesions of apposition may be observed.

The vaginal mucosa may show atrophic changes before there is any significant volumetric alteration. The number of layers of epithelial cells in the mucosa decreases to four or five. There is a complete disappearance of the functional layers. The cells of the basal layer appear small and their

nuclei and granular cytoplasm stain darkly. There is an absence of cellular glycogen. The vaginal reaction, accordingly, becomes neutral or alkaline instead of acid, as it was during the reproductive years. Alterations noted are:

The vaginal mucosa may show atrophy and consist of women's

Externally, the external os appears wrinkled and the mucosa is atrophic. The mucosa is atrophic and wrinkled.

and may result in infection about the vulva. The vaginal wall is restricted but when there has been previous trauma, the vaginal walls roll outward and there may be gaping of the parts. The vulvovaginal glands involute and their secretion becomes decreased.

Breasts. Resorption of fat and a loss of fullness of the breasts. The nipples decrease in size and areolar pigmentation decreases. There is a decrease in nipple and areolar pigmentation.

During the premenopausal stage, irregularities are observed in the breast lobules including cystic dilations of the alveoli and proliferation of the lobular buds. During the postmenopausal phase minute cysts are formed and there may be dilatations of the tubular system. As the hypoestrogenism increases the lobules disappear, the tubules collapse, and there is a condensation of fibrous stroma. Associated with these atrophic changes localized nodules of hypoplastic tissue and cystic dilatation of the acini may be observed.

CHANGES IN NONGENITAL SYSTEMS

The hypoestrogenism of the climacteric involves systems other than those of genital nature.

Urinary System. The urinary meatus. The lumen of the urethra and frequency of micturition and incontinence of postmenopausal women. Impaired bladder sphincter control also may result from loss of the tonic action of estrogens on the muscle fibers of the bladder sphincter. A similar action is exerted by estrogens upon the muscles of the vagina and perineum.

Anus. Vulvar atrophy also may involve the perianal region. Atrophic changes here may result in fissures of the dry perianal skin. Due to the loss of the tonic action of estrogens, relative inadequacies of sphincter action may occur.

Oral Mucosa. Oral mucosa becomes thin and atrophic. Ulcerative or other inflammatory processes may involve the buccal, lingual, and gingival mucosae due to their hypoestrogenic atrophy. The occasional coexistence of

atrophic vaginitis and ulcerative stomatitis and the relief of both conditions by estrogen therapy provides causal relationships of both to estrogen deficiency

Skin Aging of the skin may begin during the climacteric. The thin, dry, wrinkled and relatively inelastic skin of the aged is well known. It is the subject of intensive investigations,¹¹ which are designed to study the aging process itself. Goldzieher¹² has related these changes in the female to hypoenestrogenism and has reported that local estrogen injections reverse the aging process.

Adiposity. As a rule, weight is gained as the climacteric advances. The so-called "middle age spread," due to accumulation of fat in the girdle area, is well known. Padding is also common in the trochanteric and gluteal areas. A relationship of these fat pads and of a general tendency to gain weight to hypoenestrogenism has not been established.

Osseous System. The term "ovarian rheumatism" has been used to describe various arthritic symptoms which may occur during the climacteric. The joints of the knee, hand, and wrist are said to be involved most frequently. Acroparesthesias and neuralgias may be common. The relationship of these to hypoenestrogenism has not been established.

The term "postmenopausal osteoporosis" has been used to indicate osteoporosis which occurs after menopause and to infer that it is related etiologically to the hypoenestrogenism of this epoch. The spine and the pelvis most frequently are involved. We have not found that estrogen therapy improves the condition, and, therefore, we believe that it does not differ from other osteoporosis which occurs in both sexes during aging and which is due to deficiencies of the bone matrix.

Psychosomatic Changes. Some of the physical changes which take place during the climacteric may improve rather than detract from a woman's appearance. Graves¹³ once observed that "women of innate physical refinement often show a greater delicacy of lineaments as they approach and pass the menopause. Some women, especially those of a nulliparous and maiden class, acquire at the menopause an attractiveness that they never before possessed." It is well known that many women, following menopause, feel more secure, less burdened, and less tied to home than previously. Indeed, many women at this time develop an aggressiveness and an assurance which leads them to assume active and vigorous roles in society and in business.

Psychosexuality. The psychosexual alterations of the climacteric are conditioned, in a large degree, by the general level of health, by domestic and economic security, and by the psychic orientation to the physiologic epoch.

Cotition with normal orgasm may continue for years after the menopause until regressions of the vaginal tract impair the copulatory function. The vulvovaginal glands well past menopause may continue to secrete under sexual stimulation. It has been said that masturbation may be common even during the seventh decade and that cyclic variations in eroticism of lunar type may be described. Hamilton¹⁴ has summarized some of the psychosexual symptomatology as follows:

"Many women find that, with the subsidence of the menopause, there comes a withdrawal of interest from environmental concerns, a dreary sense of dissatisfaction, preoccupations with gastrointestinal, and other bodily functions which may pass over into a more or less serious morbid melancholy

and anxiety, sexual frigidity, a general egoistic outlook upon life and resultant unsympathetic, selfish, querulous attitude with persons towards whom they formerly sustained a more wholesome relationship. They are easily offended, feel slighted when there is no adequate objective grounds for this reaction, develop a host of petty grievances, spend a good deal of emotion on self pity, and look for scapegoats on to whom they can project their inner dissatisfactions."

Metabolism. Estrogens, and the other sex steroids, have metabolic actions. It is likely that climacteric hypoeestrogenism may result in metabolic disturbances. Among the metabolic actions of estrogen are the promotion of a positive water balance, the promotion of a positive sodium balance, stimulation of nitrogen metabolism, and, perhaps, a favoring of the storage of glucose.

THE CLIMACTERIC AND PREEXISTING DISEASE

Climacteric hypoeestrogenism may have diverse effects upon preexisting disease.

Obstetrical Trauma. It is not unusual for previous obstetrical injuries to become symptomatic as climacteric hypoeestrogenism increases. Until this time, muscles remain tonic and, because of efficient estrogen levels, they may have been able to compensate for lacerations, particularly those involving the bladder base, the rectal sling, and fibers of the sphincter ani. As ligamentous structures become weak and atrophic and the muscles become hypotonic, cystocele and rectocele may bulge at the introitus, producing pressure symptoms, a bearing down sensation, and urinary and rectal difficulties. Prolapse of the cervix and of the uterus may occur. At times there may develop extreme elongation of the cervix with ulceration due to exteriorization.

These relaxative conditions may be prevented by good obstetrical care and by the proper repair of obstetrical injuries. Treatment of these may be palliative with pessaries but the curative treatment is surgical.

Cervical Disease. Cervical erosions tend to disappear following the menopause. They are encountered infrequently after this time.

Cervical carcinoma has been thought by some to progress more rapidly when it occurred during the reproductive epoch. The inference was that the relatively high estrogen levels enhance its growth. There has been recently a reversal of this opinion. No proof has been provided that the climacteric exerts an antagonistic effect upon this form of carcinoma. Although the average age of incidence of cervical carcinoma is generally believed to be approximately that of menopause, its incidence is much less in women who have had menopause or have been castrated than in women with active or fairly active ovaries.

Irradiation is the most important phase of treatment of cervical carcinoma. Many gynecologists, however, are following this with surgery, employing the Wertheim operation. Excellent results are being reported.

Uterine Disease. Myomata and fibromyomata share in the climacteric

uterine disease. It is often overlooked that the fibromyoma may be associated with an endometrial carcinoma. A careful curettage should exclude this possibility.

Endometriosis is relieved by climacteric hypoestrogenism, since the endometrial implants become asymptomatic when there is no longer cyclic estrogen to stimulate them

The greatest incidence of endometrial carcinoma is between the ages of fifty and fifty-nine years. This means that the condition originates in the majority of women after the climacteric. There is no evidence that the climacteric *per se* has any influence on preexisting endometrial carcinoma. Furthermore, there is no clear-cut evidence that estrogens produce endometrial carcinoma.

The possibility of an endometrial carcinoma should be considered when there is abnormal uterine bleeding, particularly prolonged bleeding, intermenstrual spotting, or too frequent bleeding, in a woman approaching the menopause. It should be regarded as likely that there is an endometrial carcinoma when there is postmenopausal bleeding. A careful curettage should rule out this condition. Treatment of endometrial carcinoma is by irradiation and subsequent panhysterectomy with bilateral oophorectomy.

Premenstrual and Menstrual Symptomatology Diverse abnormal symptoms associated with the premenstrual and menstrual phases of the cycle commonly are relieved by the climacteric. These include premenstrual tension, dysmenorrhea, menstrual edema, and so called menstrual migraine. Despite this fact, it is not good therapy to induce the menopause to cure these various symptoms. Oftentimes these patients react very adversely to an induced climacteric.

Cyclomastopathy The various breast conditions, which are grouped under the term cyclomastopathy and which include mastodynia, adenosis, and cystic disease, are improved by the climacteric; they apparently require functional ovaries for their discomforts.

Mammary Carcinoma Breast carcinoma causes about 20 per cent of cancer deaths in women. The majority of patients are in the premenopausal stage of the climacteric.

There has been a belief that estrogens stimulate the growth of breast carcinoma. Accordingly, some clinicians have advised and practiced castration. It has been reported that the primary lesion as well as pulmonary and osseous metastases often recede following surgical or roentgenologic castration. The present consensus, however, is that the improvement is of a temporary nature because estrogens from other sources (adrenal cortex) intervene and vitiate the effects of castration. There are different views, however, some clinicians have reported regression of breast carcinoma following treatment with synthetic estrogens.

HYPOESTROGENISM AND PREDISPOSITION TO DISEASE

Climacteric hypoestrogenism may predispose to disease in various organ systems.

Vulvitis Atrophy of the vulva, by lowering local tissue resistance, may predispose to irritation, pruritus, and infection. Involvement of the urethral meatus and the periurethra may result in adhesions which may cause stricture.

These symptoms
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locally for the vulvar atrophy and the pruritus. Dilatations of the urethra may be necessary to correct stricture.

Leukoplakic Vulvitis and Kraurosis Vulvae. These conditions begin most commonly on the labia, about the clitoris, or on the fourchette. The skin appears dry, shiny, and white and feels thickened and like parchment paper. Considerable pruritus is associated. These conditions are precursors of carcinoma since it develops in about 25 per cent of patients. Although some relief of local symptoms may be obtained from estrogenic ointments (0.5 mg of diethylstilbestrol to 4 gm of ointment), the treatment should be excision or vulvectomy as a prophylaxis against cancer. After vulvectomy, there may be local recurrences; estrogen ointments are very valuable in the treatment of these.

Carcinoma of the Vulva. As a rule, this is a disease of postclimacteric women, the average age of patients when first seen is sixty years. It has been said that carcinoma of the vulva is perhaps "the most badly misdiagnosed and poorly treated condition in the entire field of malignant disease" (Parsons).¹⁵ Its occurrence in atrophic vulvae suggests a possible relationship to hypoestrogenism. Treatment is complete vulvectomy with a block dissection and removal of the inguinal glands.

Vaginitis. Atrophy of the vaginal mucosa renders it more prone to infection. Accordingly, organisms of relatively low virulence may produce "senile vaginitis." This is characterized by superficial ulceration and erosion and spreads by coalescence. Fine plastic adhesions may form. The condition may impair coital function and result in traumatic bleeding.

A specific treatment is effected by estrogens which restore active vaginal proliferation and cornification, and vaginal acidity. These changes are temporary and regress when treatment is stopped but the infection usually does not recur. An effective treatment is the nightly insertion of a vaginal suppository containing 0.5 mg of diethylstilbestrol for four to six weeks, followed by a vinegar douche each morning. Symptomatic relief usually occurs within seven to ten days.

An acute gonorrheal infection may attack the atrophic vulva and the vagina of a postmenopausal woman in the same fashion as in childhood.

Even when there is no vulvitis or vaginitis, local estrogen therapy may constitute valuable preoperative and postoperative measures in the postmenopausal woman who requires plastic vaginal surgery. The purpose of this treatment is rendering tissues less friable and favoring *per primam* healing. A vaginal suppository containing 0.5 mg of diethylstilbestrol is inserted nightly for ten days prior to operation. During the same time, 4 gm of ointment containing 0.5 mg of diethylstilbestrol is applied daily to the vulva. This treatment may be continued for ten days after the operation.

Cervical Obstruction. Atrophy of the cervix and apposition adhesions may result in stenosis of the endocervical canal, poor drainage of the uterus, and subsequent hydrometra or pyometra.

Carcinoma of the Cervix. It is a common cause of death in women after castration or the climacteric.

Endometritis. Apposition adhesions may form in the atrophic endometrium and local ulcerations and slight bleeding may follow. These adhesions may harbor local pockets of pus, and, thereby, produce local pyometra.

Curettage permits a diagnosis. Endometritis constitutes a small but definite cause of postmenopausal bleeding.

Endometrial Carcinoma No proof has been offered that hypoestrogenism predisposes to endometrial carcinoma, although the average age of patients (fifty eight to fifty nine years) at the onset of the symptoms suggests its possible association.

Ovarian Tumors Involution of the ovaries does not preclude neoplasia

may activate ovarian elements which were dormant when eugonadotropism existed. Indeed, granulosa cell and theca cell tumors may result from this activation. (Thirty per cent of granulosa cell tumors occur after menopause whereas theca cell tumors are more common after menopause.) The fact, however, that these tumors do occur before menopause indicates that hypergonadotropism is not necessary for their genesis. Also, the fact that virilizing tumors (arrhenoblastomata and adrenal cell tumors) do not occur commonly after menopause is against the theory of hypergonadotropic genesis, which would seem to be favored for androgenic elements by the suppression of cortical activity of the ovaries (hypoestrogenism).

Oral Infection Atrophy of the oral mucosa may predispose to ulceration and infection. These infections respond well either to local or to constitutional estrogen therapy. Dosages should be kept small and similar to those for "symptomatic climacteric" in the postmenopausal women. Mouth washing with solutions of estrogen (as liquid premarin, a teaspoonful in a third of a glass of water, three to four times a day) may be effective.

Diabetes Mellitus Hypoestrogenism and its related pituitary hyperactivity probably predispose to diabetes mellitus, thereby explaining the climacteric peak of incidence of the disease. Estrogen therapy improves climacteric diabetes.

Hypertension The instability of the vegetative nervous system during the climacteric frequently results in lability and elevation of the systolic blood pressure. It is unlikely that this "climacteric hypertension" predisposes to or is etiologically related to essential hypertension or hypertensive cardiovascular disease (Chapter 30).

Epilepsy The appearance of epilepsy during the climacteric does not warrant the assumption of etiologic relationship, save that the neurovegetative crisis serves as a "trigger mechanism" to a latent epilepsy.

Involutional Melancholy Despite the comments of some clinicians that there is a specific etiologic association between hypoestrogenism and involutional melancholy, consensus denies this and, as a corollary, denies any specific curative value of estrogen therapy in this condition. (See Chapter 17)

CLINICAL COURSE

The symptoms of climacteric women vary in characteristics and in intensity. Only a minority of these women experience distressing symptoms.

Alterations of Bleeding Commonly the intervals between flowings are longer or episodes of bleeding are missed. The flow ordinarily decreases in duration and in amount. Rarely does the menopause come abruptly. As a rule, irregularities occur for six months to a year before the actual menopause.

Too frequent or prolonged bleeding is not normal. When this symptom occurs a thorough gynecologic examination, including speculum inspection of the cervix and curettage followed by a careful pathologic study of the scrapings, should be done. Malignancy should be regarded as a distinct possibility until it is eliminated by these studies.

When there is a return of uterine bleeding after the menopause the existence of malignancy should be assumed until curettage rules it out. When curettage reveals active endometrial proliferation in a postmenopausal woman, there is a good possibility of granulosa cell tumor when previous estrogen therapy can be ruled out. One of the most common causes of postmenopausal flowing at the present is overtreatment with estrogens.

Subjective Symptoms. It is likely that 70 to 90 per cent of normal, healthy women pass through the climacteric without any symptoms severe enough to upset general health or domestic and social activities. Some women will have no unusual symptoms at this time.

The majority of women, however, have mild and rather vexing symptoms which are related chiefly to the vasomotor system and to the psyche. The symptoms include hot and cold "flushes," insomnia, emotional instability, a predisposition to confusion and disconcertion, and fatigue.

The duration of symptoms varies. The symptoms may last only a few months or a year or more. These commonly manifest cycles of severity and spontaneous improvement.

Headache is a fairly common symptom. Hyperfunction of the pituitary has been assigned as a cause for these headaches. No proof of this has been provided. Roentgenologic therapy of the pituitary is of doubtful value and roentgenologic therapy of the ovaries, in order to make the climacteric complete, usually aggravates the headaches. It is well to consider other causes of headaches before diagnosing "climacteric headaches."

HYGIENE AND TREATMENT

Since the climacteric is a physiologic epoch, its hygienic aspects should not be overshadowed by therapeutic considerations. Indeed, only a minority of women require treatment at this time.

Hygiene. Hygienic measures should include the following: there should be an orientation and indoctrination to this phase of life, unnecessary damage or sacrifice of ovarian function should be avoided, local gynecologic diseases, foci of infection, functional and constitutional disturbances, and metabolic errors should be corrected, responsibilities and activities should be scaled down moderately, and, if possible, a congenial environment should be provided. Periodic medical examinations serve two important functions: they permit close check on the patient's physical condition and they offer incidentally a good opportunity for counsel between the patient and the physician.

Preventive Treatment. There is no true preventive treatment, save an avoidance of unnecessary sacrifice of ovarian function.

The involutional (germinal and endocrine) alterations of the aging ovaries may not be forestalled. The subsequent hypoestrogenism, however, may be prevented indefinitely by substitutional therapy with estrogens.

Full Substitutional Therapy. The clinical characteristics of the climacteric, save the sterility, may be avoided by full substitutional therapy with estrogen, employing daily dosages equivalent to 1 to 3 mg. of diethylstilbestrol. If this

therapy is given cyclically—twenty days of treatment and ten days of no treatment—not only are the regressional changes avoided but cyclic and essentially normal episodes of withdrawal bleeding may be effected throughout the remainder of life. This is due to the fact that the genital organs, other than the ovaries, maintain their normal responsiveness to their proper stimuli. This therapeutic regime, however, is *not* justified at the present state of our endocrine, gynecologic, and geriatric knowledge. It doubtlessly is fraught with many apparent, as well as unforeseen, hazards. We understand the rationale of estrogen therapy during the climacteric not to be prevention of the sequelae of ovarian senility but to be rendering less critical and less severe the subjective symptoms which are associated with this involution.

Much current estrogen therapy is so excessive that it simulates full substitutional treatment. Postmenopausal flowing, resulting from estrogen therapy, is a too frequent occurrence. The psychic and emotional trauma occasioned by this bleeding negates the therapeutic indication, that is, symptomatic palliation, and it frequently impels a physician to recommend a diagnostic curettage lest a carcinoma be overlooked.

Other distressing symptoms which may follow excessive estrogen therapy during the climacteric are these: there may be frustration and delay of the process, premenstrual like abdominal cramps and breast pains may occur, vaginal irritation and pruritus may be experienced, and a generalized edema may develop.

What we term "shock treatment" may prove as troublesome as excessive treatment. By "shock treatment" we mean the practice of giving injections of relatively large amounts of estrogen at irregular and usually at infrequent intervals, depending upon the wishes of the patient and the whims of an office nurse. We regard this "shock treatment" as an ideal formula for producing a chaotic endocrine system and for assuring a prolonged climacteric.

Palliative Treatment. When palliation of severe climacteric symptomatology is needed, estrogens afford specific treatment and should not be denied the patient. Estrogens are far superior to barbiturates, bromides, benzedrine, and other sedatives and stimulants. The dangers of these drugs are numerous and well known, and there seems little justification for exposing a patient to these dangers when the natural hormone, estrogen, is available and is specific.

The estrogen of choice for treatment is the natural hormone and not the synthetic product. The reasons for this statement are (1) Natural estrogens are well tolerated. (2) They give a sense of well being. (3) They provide beneficial metabolic actions.

Estrogen therapy should be orally administered. Repeated office visits for unnecessary and expensive injections constitute a poor psychologic approach to the climacteric. The schedule of estrogen therapy should be compatible with a continuance of the involutional process, it should not upset this process.

Estrogen should be given cyclically: twenty days of treatment and ten days of withdrawal of treatment. When the premenopausal woman is treated, estrogen should be given during the interval and the withdrawal should coincide with the bleeding phase.

Approximate treatment schedules are given to illustrate our concepts of dosage and the scope of treatment. We prefer "conjugated estrogens," avail

able commercially as premarin—as tablets of 0.625 mg. and as a liquid each teaspoonful of which equals 0.625 mg.

(1) The premenopausal woman. Somewhat larger dosages are given than when the postmenopausal woman is treated. Two to four tablets (0.625 mg. each) of premarin are given daily for twenty days and treatment then is withheld to await bleeding. Treatment is resumed at the end of bleeding and the same dosage is employed for the second cycle.

During the third and fourth cycles of treatment the dosage is reduced by 50 per cent.

During the fifth and sixth cycles of treatment 10 drops of liquid premarin are given three times a day for twenty days followed by the usual withdrawal of treatment before and during bleeding.

(2) The postmenopausal patient. The amounts of estrogen used must be small lest postmenopausal flowing be induced.

During the first two cycles of treatment 15 drops of liquid premarin are given four times daily and these treatment cycles are followed by ten days of withdrawal of treatment.

During the third and fourth cycles of treatment 10 drops of liquid premarin are given four times daily for twenty days and followed by withdrawal for ten days.

During the fifth and sixth cycles of treatment 5 drops of liquid premarin are given four times a day and followed by withdrawal.

Geriatric Estrogen Therapy. Some clinicians advise that all climacteric and senescent women should receive small basal amounts of estrogen to prevent the ravages of age and to maintain normal metabolic and sexual functions. More information is needed before this is accepted as a general procedure. It has distinct advantages and indications in the forestalling of sexual aging in women who have had premature climacterics either of spontaneous or artificial origin.

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SECTION VIII

DISORDERS OF THE SKELETAL SYSTEM

CHAPTER 42

DISEASES OF THE JOINTS

ROBERT T. MONROE

ARTHRITIS is universal in old age. So also is the fear of it. If all signs and symptoms referable to joints are collected together under this heading it is clear that few individuals complete the first half of their lives without evidence of disorder in some of their joints, and as time goes on the evidence extends and accumulates. It is equally clear, however, that relatively few individuals suffer severely or persistently or increasingly, especially as old age sets in. Knowledge of a few simple distinguishing characteristics makes it possible to tell whether the arthropathy is one which will interfere seriously with health and activity, or one which will be merely a discomfort and a nuisance. Accurate diagnosis, therefore, is of the greatest importance in evaluating the significance and the alarm of the rheumatic maladies of old people. Precision can be attained even in long standing cases in which more than one pathologic type of arthritis is observed, since only one type of reaction is clinically active in a joint at a given time. 'Mixed arthritis' is for this reason an unnecessary and confusing term. In this chapter an attempt will be made to describe the characteristic reactions of diseases of the joints and the manner in which they will be affected by senescence.

ARTHRITIS

Types of Arthritis. A joint consists essentially of only two tissues, cartilage and synovial membrane. They respond to disease so differently that two main types of arthritis result. Cartilage has no vascular channels, hence it cannot become hyperemic or inflamed or react to known infective agents. Cartilage can only break down, so that trauma in some form and degree is the one recognized etiologic factor. This type of arthritis is called traumatic or degenerative or *hypertrophic arthritis*, or *osteoarthritis*. Contrariwise, synovia, the rather incomplete lining of the richly vascular capsule, escapes trauma easily. It responds to disease by becoming hyperemic and inflamed, these appearances usually are accounted for on the basis of bacterial invasion. The synovial type of arthritis is called infectious, inflammatory, proliferative, rheumatoid, or *atrophic arthritis*. Tissues surrounding joints are involved in the arthritic process: bone by eburnation in the first type and by inflammation and rarefaction in the second, tendons and capsule by fibrillation in the one and inflammation in the other, and bursae by calcification or inflammation. The total clinical picture, however consistent ultimately with trauma or infection, varies enormously according to the degree of injury and disease in each joint.

taken together with its supporting structures. My preference is for the terms "hypertrophic" and "atrophic" arthritis because they seem best to describe the general situation of the victim of the arthropathy. Yet there remain many rheumatic symptoms and states, which appear not to be based primarily upon intra-articular lesions, but which involve chiefly the supporting structures. These are loosely defined and poorly understood conditions called muscular rheumatism, fibrositis, tendonitis, tenosynovitis, bursitis, capsular rheumatism. They have been grouped here as a third type under the term "peri-articular fibrositis." For a fuller defense of this somewhat arbitrarily simplified classification of diseases of the joints, the reader is referred to articles in standard texts of medicine 1, 2, 3.

Senescence of Joint Tissues. The alterations which the passage of time causes in joints are poorly understood. The excellent summary of the presently known data by the late Professor Todd⁴ leaves the critical clinician with a baffled feeling of uncertainty. The changes due to senescence may be very few, or they may be many but as yet unappreciated, or they may not reasonably be distinguishable from the cumulative effects of metabolic, nutritive, traumatic, and infective agents. Hyaline articular cartilage shows some degree of wearing away in all individuals as soon as they are fully matured, although external manifestations may be delayed until middle or late maturity. This erosion is not followed by metaplasia to fibrocartilage unless inflammation due to infection or hemorrhage has permitted access of connective tissue to the surface of the cartilage. By the end of the sixth decade of life, the fibrocartilage of the intervertebral disks shows fibrillation and penetration of connective tissue, often with necrosis and calcification. Herniation of this cartilage into the adjacent vertebral plates is common. The nucleus pulposus loses water and shrinks. The synovial membrane of joints and bursae is such a simple and easily regenerated tissue that it shows no changes with age. Invariably, however, tendons and ligaments are altered. Crepitus can be palpated in the shoulders of most active individuals more than twenty-five years of age, due to splitting and fibrillation of the capsule and of the supraspinatus tendon. Later in life calcification at the bony attachments of ligaments is noted, causing irregularities (for example, on tibia and ulna) and osteophytic spurs about joints. Muscles also show some disruption of their usual architecture, bundles are distorted and fibrous tissue and collagen increase between them. Age changes in the vascularity of periarticular tissues are in dispute. Some observers deny that there is any reduction in blood supply due to age or arteriosclerosis, others believe that they have demonstrated a significant reduction. Clinical evidence fits best with a diminution. Aging processes in bone are of obvious importance to the integrity of joints, for malpositions cause different stresses and strains on cartilage, capsule, and tendons. It may be well to emphasize that few manifestations of age in joints appear late in life. Many develop in the middle years, some even earlier.

ATROPHIC ARTHRITIS

In simplest terms atrophic arthritis is a multiple inflammatory synovitis. There must be many cases of a mild or transient sort which heal with almost no residue and are of interest only as they may serve to explain a certain vulnerability to degenerative changes later on. We are concerned here with the much more uncommon form of multiple inflammatory synovitis, *chronic*

atrophic arthritis which inclines to progression or periods of reactivation for the duration of life. The etiologic agent or agents are unknown. It is perhaps most reasonable to assume that any infection may be responsible, the variable effects being due to differences in bacterial dosage, repetition, and state of the victim. In the majority of cases the onset occurs between the ages of twenty five and fifty, women being affected thrice as often as men. Its identification depends upon the recognition of certain dependable characteristics in the joints and in the patient.

Any joint which has a synovial lining may be affected. Many joints are attacked in the first bout and others may be attacked in subsequent relapses. When only a very few are diseased, a frankly septic or specific (gonorrheal) arthritis is probably present. The number of inflamed joints is not a guide to prognosis; however, any more than the number of enlarged lymph glands is an index to the severity of Hodgkin's disease. The joints most often affected are the *peripheral* ones, such as fingers, wrists, elbows, ankles, knees. Trauma, use and weight bearing do not affect the selection of sites, although they may intensify the destruction once it has been started. A curious tendency to *symmetrical involvement* appears sooner or later, for example both wrists, both knees, or the same fingers on each hand.

Active Stage. In the active stage the inflamed joint is swollen to the capacity of the synovial space and that of the surrounding bursae, so that it presents a smooth, often fusiform appearance. It is soft, doughy, or tense according to the amount of fluid in and around it. It is tender to touch and painful to move. *Function* is thereby limited. Motion may be accompanied by a leathery, sticky rub. The muscles which control the joint undergo *atrophy* very early in the course of the disease. The wasting often appears to be more than the degree that might be attributed to disuse; some of it remains permanently despite fair healing and resumption of use. A rather fine tremor may be found during the active stage. The skin about the joints also atrophies, appears pallid, thin, warm or cold, and drips with sweat. In the quiescent periods it becomes dry but remains somewhat thin and is easily traumatized. Radiography discloses rarefaction of the apposed bone ends, and this also tends to be permanent. Deformities of the joints arise from proliferative processes throughout the capsule and down through the cartilage, and from weakening of the tendons and muscles. Ankylosis, subluxation, hyperextension, flexion and deflection follow in greater or less degree.

The patient shows the usual *systemic manifestations* of a long standing infection. A low grade fever may run on for weeks or months. The disease may progress without fever, but as a general rule, the higher the temperature the more active the disease and the brighter the outlook for early abatement. The pulse is rapid as long as there is active inflammation. Young patients look much like those with rheumatic fever, and about 10 per cent of the cases show valvular heart lesions. About half of the patients show loss of weight. Where the weight is maintained or increases there is an appearance of pudgy obesity. Reactive depressions are quite common, as would be expected in any situation which entails pain, wasting, disability and fear. A moderate hypochromic anemia often is present. The white blood cells are increased slightly in number in the more active cases, in indolent cases their number is normal or reduced. The sedimentation rate of the red blood cells is increased.

Inactive Stage. Considerable clinical acumen is required to tell when at

rophic arthritis is active and when it is quiescent or healed. Yet naturally it is necessary to know this if treatment is to be directed intelligently. Absence of fever and leukocytosis and gain in weight are not reliable indications that the process in the joints has stopped. Pain may be caused by the synovitis but it may also be due to mechanical stresses or to fatigue, or it may be an expression of poor morale. The best laboratory sign of activity is an increase in the rate of red blood cell sedimentation. A persistently rapid resting pulse rate (more than 80) is just as good a guide, barring the presence of coincident heart disease. Fluid which is clearly inside joint spaces must be taken as evidence of activity. Fluid in bursae and tendon sheaths means a lesser degree of activity and still lesser traces are suggested by stiffness which is quickly erased by use. Pudgy, soft swellings between joints are not signs of activity. Tremor of atrophied muscles and sweating of the skin over involved joints indicate activity.

Course The course of the disease is one of exacerbations and remissions over a period of many years. There are great individual variations. The important factors are (1) the dose, nature, and frequency of exposure to infective agents (frankly septic joints heal quickly and permanently with a quantum of irreparable damage and no systemic effects, lesser grades create less effective bodily reaction and tend to smoulder for many weeks and to flare up with ease), (2) the treatment, and (3) the level of health and physical fitness maintained between attacks. My impression is that the recurrent and progressive nature of the disease could be stopped forever if one could treat adequately an early attack, guide the patient toward vigorous fitness, and take every suggestion of relapse seriously over a period of five years. The average patient, however, does not report to a physician until several attacks have convinced him that he is in for trouble, treatment is often inadequate in conception and execution, and supervision of convalescence is sketchy—so that the picture called to mind by "chronic arthritis" is that of some degree of multiple joint disorder, frequently recurring pain, weakness and disability, a low level of endurance, and a personality and career twisted by present and anticipated slurs and arrows from an outrageous fortune. As the years add up to old age, this average patient poses many perplexing problems for his family medical adviser.

Cases of atrophic arthritis with first attack after the age of sixty are rare. In a series of 300 cases, seen over a period of ten years, I found that the arthritis began in the seventh decade in only 4 per cent (five cases) of the men and 10 per cent (twenty-two cases) of the women. Two of the men had acute gonorrheal arthritis, one had cancer of the lungs, one had cancer of the bladder, and one had been in a state of severe malnutrition. The women likewise had outstanding abnormalities in their physical or social environment at the time when their joints first showed disease. This is in contrast to the onset in younger people in whom it often is very insidious and unaccompanied by any other outstanding event. These new cases in old people tend to be severe, with marked atrophy of muscles and skin, loss of weight, and disability beyond observed cause. Fever and tachycardia are found as in young people, but there is a more profound hypochromic anemia and a tendency to leukopenia.

Treatment of Active Atrophic Arthritis The two essential features of therapy from start to finish are rest and exercise. *Rest in bed* is basic for any

generalized inflammatory process. Yet the arthritic shuns it. It seems to him an admission of defeat and an acceptance of invalidism, for inactivity seems to increase stiffness. He must be instructed that most of the stiffness is due to distention of the tissues in and around the joints with the products of inflammation. Such distention can be reduced by active or passive motion but only at the risk of spreading and intensifying the disease just as motion is harmful to broken bones or pulmonary tuberculosis. It is reduced naturally when the arthritis abates, so that stiffness in the morning is a reliable sign of active disease and lessened stiffness after rest is a sign of improvement. It takes only a short time to prove the correctness of this view for in that short time the patient begins to benefit from a lightening of the load of fatigue that was oppressing him, his morale improves, his stiffness is better or at least no worse, and from then on his intelligent cooperation is secured.

The duration of bed rest is measured by the time required for the physical signs and the sedimentation rate of the red blood cells to return to normal. It is usually a matter of six weeks or more. Hospitalization accomplishes results quickest because it imposes the program with least argument and in a good environment. However, hospital beds are very expensive, they are rarely available for such long periods of time, hospital staff members are not sufficiently interested in chronic disease, and elderly patients often feel so homesick for their usual haunts and habits that care in a hospital may do more harm than good. So rest must be supervised at home in most cases. The bed should be a single one with a firm mattress, and in a room apart from other members of the family. Provision should be made for discharging toilet duties with the least effort by the patient. Hours for visitors must be regulated. Quiet periods without reading or talking or listening should be enforced in the morning and afternoon, and lights should be extinguished early at night. These are trifling suggestions, perhaps, but important nevertheless, and not implied in the sterile order to "stay in bed." That accomplishes nothing unless it is translated in minute detail to the patient, his family, and his friends, and in terms of his particular circumstances and environment.

Special attention must be given to secure rest for the affected joints in as optimal a position as possible. Well made plaster casts for elbows, wrists, or knees allow a degree of relaxation and freedom from spasm that may not be accomplished otherwise. For several periods daily the patient should lie flat—the head without a pillow, the arms abducted, elbows extended, wrists in line with the forearms, fingers outstretched, hips and knees extended, the feet at right angles to the legs. Interference by bed coverings is lessened by use of a cradle or by a board or a pillow at the foot of the mattress. Long hours of stagnation in crouched and awkward positions are to be avoided. Modifications of this attitude obviously are necessary for comfort at times. Reasonable efforts to use it, however, are of the greatest benefit in lessening the development of fixed deformities in joints and in alleviating symptoms due to mechanical strains from awkward postures.

Exercises are just as essential in the healing process. They help to speed up absorption of inflammatory exudates, to lessen atrophy of muscles, and perhaps to lessen decalcification of bone. It is not wise to delegate the responsibility of prescribing them to a physiotherapist, for well-trained technicians are not often available. The exercises are very simple in conception and

easily learned. The patient is encouraged to move each affected joint slowly and steadily, without jerking, to the point at which joint pain begins, but never through pain, for their presumably inflammatory tissues are torn and repair is impeded. A series of such active motions is repeated two or three times daily. The time to begin them is on the first day the patient is seen. No matter how acute and painful the arthritis may be, experience teaches the gain that results from even slight motion of sick joints. Passive motion to increase the range of joint function is hazardous; by unskilled hands it can do great harm. Exercise of unaffected, normal joints should be prescribed also daily and from the beginning. Deep breathing aids pulmonary ventilation, stretching of the back, arms, and legs improves morale and comfort in bed, and possibly lessens the risk of thromboembolic accidents.

The program of rest and exercise is continued until the arthritis is arrested and function is restored. As signs of recovery appear, the exercises are increased cautiously, and the time for visitors and ordinary amusements is lengthened. Monotony can be evaded by inventing safe games to supplement exercises and by performance in time to music. Weeks, even months, are well spent in getting the patient out of bed and back to normal activity. Stiffness, a rapid pulse after rest, and morning fatigue are signs of a relapse or a too hurried schedule—signs which it is wise to heed. A morning rest period may be abandoned in half a year, but the afternoon nap might well be made a permanent habit. Every attempt to neglect exercise, shirk rest, or indulge in activities which interfere with the program must be fought. Otherwise disheartening losses of joint function return, or avoidable deformities become established. This advice is the same as that which is given to young patients. Old people have an equal right to it. To cut treatment of the old according to life expectancy or economic usefulness or even their own demands is to invite complete failure. Nature does not do as good a job in producing spontaneous remissions in the old as she does in the young.

The *diet* should be a full normal one—a pint of milk, fresh fruit, green and yellow vegetables, meat or fish or poultry or eggs every day, bread, potatoes, pastry, and sweets in rather less than customary quantities. Sub-optimal eating habits are very common, especially in old people, and they are hard to change. It is worth while to attempt it. Meanwhile it is fair to use a multiple vitamin pill daily. An excess of coffee or tea prevents relaxation. Three meals a day with nothing to eat between them aid in restoring appetite and discipline. Some old people feel weak unless a fourth meal is provided. However, the tendency to humor finicky tastes, or to supplement half cleared trays with rich drinks or snacks, is to be frowned upon. A gain in weight is a sign of improvement only when it is not made up of fat. Indulgence does not foster a fighting spirit.

Care of the bowels has received too much attention. There is no evidence that constipation harms joints or that diarrhea helps them.

Heat is helpful. Old people are sensitive to cold and those with active inflammatory arthritis still more so. Comfort is promoted by the use of flannel sheets or night clothing and by protection from draughts and a chilly atmosphere. Heat to inflamed joints for a short period daily before exercising promotes hyperemia. This increases circulation and the dissipation of fluids which contribute to stiffness, and it improves the performance and results of exercises. A hot-water bottle or electric heating pad or a simple bed cradle

with an electric light bulb appears to accomplish as much as more expensive apparatus. One must be careful not to overdo baking, for atrophic old skin burns easily. Liniments are much in favor with the aged. They do no harm if they are not applied with vigorous massage, and their odors bolster faith.

Pharmacotherapy has little to offer for the average case of atrophic arthritis. Penicillin or sulfadiazine is indicated in full doses in patients with frank infections in the joints (septic or gonorrheal arthritis) and frequently it proves worth while to give them to arthritic patients when they first come down with infections of the upper respiratory tract, when they face dental extractions, or when a new accession of fever, leukocytosis, and synovial effusion suggests that they are in for a flare-up in arthritis. They appear to be of no value in cases without fever or leukocytosis, which means most of the cases most of the time. In such cases sulfonamides seem to enhance a tendency to anemia, leukopenia, and anorexia, and obstructive situations in the kidneys, especially in old people. Small doses of salicylates promptly decrease fever and alleviate pain in febrile cases. I have an impression that middle-aged and elderly individuals cannot take large doses of salicylates over a period of weeks. This is based on several experiences in which a slight persistent fever, aching malaise, and loss of appetite cleared up the day after salicylates were discontinued. Perhaps they are used best in moderate doses for definite purposes, for example to erase pain which interferes with sleep. More powerful analgesics such as morphine and demerol, are not indicated. Intractable pain, which calls for them, must lead to a search for malignant disease. Gold salts are now widely used in research clinics although their mode of action is not understood and the incidence of toxic reactions is rather great. The toxic effect of gold in kidneys, skin, and liver appears to increase with the age of the patient. At present the safest stand to take is that the chance of improvement in the joint disease from the administration of gold is outweighed in old people by the probability of untoward side effects. The general pessimism as to the efficacy of all other drugs by any route—sulfur, vitamin D, vaccines, and so on—seems fully justified.

Treatment of Recurrent Active Cases These make up the vast majority of cases of atrophic arthritis found in old people.

Severe. Fortunately the complete arthritic cripple, helpless, emaciated and bedfast, with distorted, contracted joints, unable to move or feed or care for himself, is rare. One would like to feel that such a horrible state always is to be avoided by proper care, but there seem to be a few cases of such malignant proportions that nothing avails. These poor creatures have little chance or desire to live into the seventh decade of life. Intercurrent infections and malnutrition close the scene much earlier. Occasionally one such patient does linger, a monument to the devoted care of his family and nurses. Therapy has little to offer, although in younger patients reconstructive orthopedic surgery performs limited miracles.

Moderate Lesser degrees of crippling, due to repeated assaults upon the joints, are not so uncommon as they should be. Here, however, something can be done, for the ravages upon skin, muscles, and bones are not so great. Moreover, the occasional interludes of freedom have preserved an approximation of mental health. The pathetic assertion that if you cannot do any thing for the joints you can take care of the patient who has them, has definite point here. Often one is amazed at the results which can be obtained by the

intelligent use of available hobbies, by persistence in bed rest and exercises. Hopelessness can be replaced by the conviction that, if normal life is not possible, some portion of it is better than none at all. Objective interests can be fostered. Nutrition can be improved. Fatigue can be avoided. Means of locomotion can be explored. Not infrequently it turns out that the pain and malaise and discomfort that were attributed to active joint disease are in reality due to misuse or inactivity, or that they were in the foreground of consciousness because nothing else was there. Such sublimation, if so it be, occurs spontaneously due to religious conversion or to some unusual interruption in the routine of life which calls imperiously for more effort on the part of the patient. More often it must be won slowly by a persistent, ingenious physician and a cooperative courageous patient.

Amyloid disease as a sequel to the malnutrition of chronic infection is worth more consideration than it has received. Since the first edition of this work the author has had two cases. Both were in men, one fifty-eight and one sixty-eight, each had had a moderately severe, frequently active arthritis for many years. Within a space of two years, the younger had five gross hemorrhages from stomach and bowel, the older had two. In both several radiologic studies were normal, the younger had a thorough abdominal exploration with negative findings. The Congo red test showed complete retention of the dye in each. Since that time each has done well as to general health and absence of bleeding, on a high protein high vitamin diet.

Mild. Most instances of long standing atrophic arthritis in individuals over sixty years of age are mild. The deformities of the joints are of minor degree and the general health has been fairly well preserved. Sometimes it would seem that the original and subsequent infective processes must have been slight, and that an excessive price in joint destruction has been paid because of inattention to rest or insistence on unnecessary or improper use. Such joints in old age show sharp osteophytic spurs, the atrophied muscles and tendons are sensitive to trauma, and the thin, dry skin bruises easily and is poorly supplied with blood. Treatment here is largely a matter of what the physician can devise or the patient will accept. The patient is fully aware of the fact that his joints cannot be restored to normal, but he may not realize that he accepts more pain and disability than are necessary. A long daily acquaintance with his trouble generates in him, sooner or later, an attitude of neglect and of resignation to the clearly foreseen penalties for overdoing. The harsh requirements of rest, exercise, and nutrition become boring. Escape is sought in pleasure, in inaction, in the delights of the table, and a martyr's mantle is put on to cloak these sins and to show why such a sufferer should not be expected to bear his share of the family's burdens. Or the harder headed, those with ambitions and responsibilities, attempt to go their ways stoically, regardless of their joints. Either attitude is a formidable barrier

orthwhile in spite of the limitations of atrophic arthritis, but the former is more likely to be fairly mild or transient. The other is that minor changes of habits and schedule are rewarded far beyond expectations.

Strumpell-Marie Type. Patients with this severely crippling form of atrophic arthritis of the spine, hips, and shoulders rarely live into old age. Where the costovertebral joints are involved, a fixed thoracic cage results

Far too frequently, marked kyphosis occurs from failure to maintain a straight spine by prolonged bed rest in the active months of the disease. Respiration is thereafter accomplished by movement of the diaphragm alone. Of the sixteen patients with Strumpell-Marie arthritis seen in my clinic over a ten year period, only two had reached the seventh decade of life. One of these (and four others in their fifties) succumbed to pneumonia and heart failure (cor pulmonale). The other remained hale and hearty at sixty eight perhaps because his spine is as straight and as rigid as a broomstick.

HYPERTROPHIC ARTHRITIS

Degenerative changes in the joints are demonstrable in every individual well in advance of his sixtieth year. They increase rather more slowly as old age sets in, but they do not lessen or disappear. In a sense they are a measure of the inability of cartilage fully to repair itself after injury. The injuries may be clearly recognizable in a past history of occupational or sports hazards, imperfect alignment after fractures or incompletely healed sprains, or they may be the easily overlooked but frequently repeated injuries incident to ordinary life and play, as the strain upon weight bearing due to years of obesity or to static deformities. Very often convincing evidence of trauma is lacking. Perhaps such cases suggest the postulate that cartilage has a special vulnerability to degeneration on an hereditary or abiotrophic basis, and that this quantum of hypertrophic arthritis is an unavoidable part of the process of senescence.⁵ Other explanations based upon infections, metabolic or endocrine disorders have to be discarded at present for lack of experimental and pathologic proof.

The disparity between the signs and symptoms of hypertrophic arthritis is always a matter of comment. One person, middle aged, may have considerable changes in his joints.

from them, and another may have no synovitis present. A physical or roentgenologic study of the joints may reveal changes except in the occasional case. Here hemorrhage has taken place or a bit of cartilage has crumbled off and lies free in the synovial space. Pain therefore, does not arise from within the joint structures. Hence it is proposed to abandon the term "arthritis" for this form of arthropathy and to substitute "degenerative joint disease." The symptoms result from spasm of the muscles which support the joints, or from sprains of their tendons, or from pressure on the periosteum by osteophytic spurs on the joint margins, or from pressure on dorsal nerve roots by nearby spurs.⁶

In the average case only one or a few joints are the site of symptoms although many others show typical changes. The symptoms usually can be related to use, either in bearing weight or in doing work. For example, in the spine, the knees, and the right arm in right handed individuals. Wrists and ankles, curiously enough, are almost never involved. Symmetry is not a feature as in atrophic arthritis.

bursae and tendon sheaths are more common, but they seem sufficiently independent of the degenerative joint disease to warrant their inclusion in the third form of arthritis which is discussed in a later section. Function is maintained at full range, or it is limited by marginal overgrowths, not by spasm or edema. Ankylosis is seen sometimes between spurs along the vertebrae or in severe hip disease. It is not a feature of the average case, since its precursor, inflammation, is absent. When crepitus is palpated or heard it is a gritty, crunching affair, quite distinct from the leathery rub of the rheumatoid joint. The skin over the joints is not atrophic or unduly warm or moist. Muscle atrophy is absent or slight, and there is no tremor. The bone ends show no decalcification, on the contrary, their cartilaginous surfaces are eburnated.

The distress which arises from a hypertrophic joint is of many sorts. There may be a nondescript discomfort or a steady ache as in static strains. There may be sharp, severe pain with sudden trauma. Paresthesias, numb tingling, burning, paralyzing sensations, suggest pressure such as occurs in cervical spine involvement or in the ends of fingers swollen with Heberden's nodes. Pain may be referred from its point of origin to a nearby area to the deltoid insertion in disease of the neck or shoulder joint, or even to another joint as pain in the knee from disease in the hip. Stiffness may disappear after a few efforts at limbering up. In contrast to atrophic arthritis, the pain and stiffness of hypertrophic arthritis increase toward the end of the day and abate after a night's rest.

The general health of the patient with hypertrophic arthritis is good. It has to be to permit him to accumulate the wear and tear which cause his symptoms. There is no sense of malaise from this arthropathy, no infection which appears to bear an etiologic relationship, no increase in temperature or pulse rate. Laboratory studies reveal no anemia or leukocytosis and no change in the rate of sedimentation of the red blood cells.

A brief survey of postural errors may be of help in the detection of symptoms associated with degenerative joint diseases. The anterior or transverse arch of the foot is said to be flattened when there is pain at the base of the middle toes and a callus on the ball of the foot. The long arch of the foot is pronated when the astragalus sinks between the calcaneus and the navicular, this stretches the plantar capsule and tendons and causes pain in the sole and along the outer edge of the tibia. It may result from a habit of walking with the toes pointed outward, or it may suggest hip disease, one of the first signs of which is external rotation. In time it strains the knees, for most individuals who toe outward spring and knock the knees. High, narrow heeled shoes probably account largely for the prevalence of these static difficulties in women. They force an undue amount of weight to be borne upon the balls of the feet and encourage a rolling motion of the foot on walking. The body is tilted forward, so that the erect position is maintained by throwing the chest backward, thus increasing the lumbar lordotic curve.

Knock knees and lumbar lordosis are also a characteristic accompaniment of obesity. A protuberant abdomen pulls upon the lower back and causes a counterpull in the dorsal spine, when the patient tries to stand up straight. The habit of sitting in an exaggeratedly upright position seems in some instances, particularly in women, to promote lumbar lordosis so that by middle and old age a "tired backache" results. Many elderly people lose height

perhaps from narrowing of the intervertebral spaces by dehydration, perhaps from osteomalacia or osteoporosis, or perhaps just from years of slouching, and the increased and rather rigid curves in the spine which follow appear commonly to cause various radiating aches and pains around the chest and abdomen. Lordosis of the cervical portion of the spine is observed frequently from various causes such as occupations which encourage a forward position of the head (chauffeurs, seamstresses, and school teachers), a habit of reading in bed on several pillows with uncorrected visual errors, or in compensation for the round shoulders of dorsal kyphosis.

An extraordinary variety of *headaches*, pains, and paresthesias in the eyes, cheeks, neck, shoulders, and arms can be traced to fatigue of muscles and tendons in the back of the neck, tender points in the occiput, spasm in the muscles of this region, and relief on resting with the spine perfectly flat make the diagnosis reasonable. A cervical rib or an unduly long transverse process on a cervical vertebra may be present. In the majority of cases these findings prove to have no clinical significance. They are noteworthy only when they produce pain and occasionally atrophy in the distribution of the radial nerve. Scoliosis, whether functional from poor posture at a desk or from a shortened leg or arthritis of the spine or imbalance of the erector spinae muscles, causes *backache* in many elderly individuals.

The *etiology of the pain* suffered by patients with hypertrophic arthritis can be found to have a mechanical basis from poor posture or from distortion of the joints in the great majority of cases (75 per cent in the author's experience). For the remainder the explanation is not so clear. Simple fatigue appears to be a factor of importance, particularly in late middle life. This is not the fatigue of muscular exertion, for it is not found in the physically fit, it is to be remarked that for a healthy state muscles require regular use. It is rather the fatigue of stress and strain, the nervous exhaustion of persons not in the habit of taking exercise, who are under pressure in business or at home, or who get insufficient sleep and relaxation. Malnutrition is also important, especially if obesity is included with its attendant mechanical faults. Occasionally one sees persons with degenerative joint changes, whose symptoms are relieved by stopping an excessive use of coffee, tobacco, or candy or by taking an optimal diet. Many women, who suffer from vasomotor disturbances for years after the menopause, also have arthralgias with their "hot flashes." Patients with myxedema or hypothyroidism are, in the author's opinion, subject to joint symptoms which can be controlled by the judicious administration of thyroid gland substance. Skeletal aches and pains are common in middle-aged and elderly patients with hypertension, arteriosclerosis, cardiac disease, and thrombophlebitis, and they may subside entirely when these vascular states are compensated.

Characteristic Joint Changes. Joints in several locations show such definitive characteristics as to make the diagnosis of hypertrophic arthritis possible at a glance. The terminal interphalangeal joints of the hands are enlarged in a hard, irregular shape. They may be grossly distorted and the distal phalanges deflected to one side or the other, or they may show only smooth nodules on the dorsolateral edges. These are *Heberden's nodes*, named after the man who in 1802 recognized them as signifying a benign form of rheumatism. They occur much more commonly and in more severe form in women than they do in men. The reason for this sex difference is a mystery.

Trauma and exposure are hardly implicated, for often they are more developed in the dowager than in her charwoman. The other finger joints may be quite normal and suppleness not decreased. In men these nodes are normally associated with similar irregularities of the proximal finger joints and the thumbs, here the relationship of wear and tear is more evident e.g., the baseball fingers and the horny handed son of toil. These changes entail some stiffness and loss of ability to perform fine tasks. Probably the majority of individuals experience no symptoms from the nodes other than a transient ache and a fear that a crippling arthritis is on the way.

Occasionally a *cystic herniation of synovial fluid* may appear, and for a time the surrounding area may be tender and red but there is no fusiform swelling and the soft bead soon subsides without ankylosis unless unwise tapping has introduced enough infection to cause a septic joint. Occasionally also there is moderate *distention of the soft tissues* of the finger tips with numbness prickling or stinging sensations. On the whole such events are practically limited to women in the menopausal period. As old age sets in, the nodes are quite symptomless, and the finger joints remain useful with a varying degree of limitation of motion from the interlocking overgrowths. Their appearance is so diagnostic that, if the patient represents them as her chief claim to rheumatism, she can be assured at once that she has no grounds for worry. It is curious that similar nodes are not found on the toes, unless bunions can be so classified.

The *knee* is often symptomatically active because weight bearing hastens its degeneration, and so frequently the ligaments are strained by use. Yet its enlargement may be quite immaterial. Palpable irregularity is noted chiefly in the outline of the patella. Effusion is rare and relatively transitory. Tenderness, when present, more often is located around the knee, where tendons are inserted, rather than precisely along the margins of the joint. Crepitus is present as a hard, gritty, snapping, sometimes audible, crunching. Function is preserved without great difficulty, for locking is not extreme, and the muscles are tense rather than atrophied. Pain and stiffness are greater after a long session of sitting than they are after lying down. Descent of stairs is more painful than ascent, because on going down the knee is loose and on going up it is guarded by tense muscles.

Hypertrophic arthritis of the *hip* has been dignified by the name *morbus coxae senilis*. This term is in error to the degree that it suggests that the affection is confined to old people, for as a matter of fact those most troubled by it range from fifty five to sixty five years of age. Once established it persists, and it is so disabling as to warrant special mention. The degenerative changes result from the head of the femur grinding down the acetabulum. The acetabulum becomes shallow and fringed by osteophytic spurs. The femoral head is worn away so that its weight bearing lines are seriously altered. The two bones may be quite tightly locked by the spurs. Both hips are affected always, but usually one is more involved than the other. Function is limited first on external rotation, later on flexion, so that the patient tends to walk with the foot everted, to lean toward the affected side, to sit with the buttocks well forward on the chair, and he has difficulty in crossing the knees and in bending over to tie his shoes. Walking is performed with less motion of the thighs than of the lower legs, for stiffness makes it difficult to lift the foot and causes fear of stumbling. There is apparent shortening of the leg if the condi-

tion depends on inability to extend or to adduct the thigh, but this is real if the femur is worn down or pushed up on the outer edge of the acetabulum. There may be severe pain in the hip on motion or torsion, as in standing or sitting, and there may be tenderness on pressure over the greater trochanter. Many cases report only stiffness and no pain. Occasionally the only pain is one referred to the medial superior aspect of the knee.

The intervertebral joints show the most striking degenerative changes. They are clearly apparent radiographically after the fourth decade of life in all people. Trauma accentuates them for they are most marked in those who use their backs most. Postural strain contributes the extra amount which is found at the two points of buckling—the lower cervical and the lower lumbar regions. Senescence must account for some portion of this universal feature. In middle life the intervertebral disks begin to lose their elasticity. This results in the shocks being borne directly by the vertebral bodies, whose surfaces become heavily calcified. The shrunken disks allow more motion to take place so that ligaments and capsules are pulled, and this constant irritation brings out the characteristic fringes of osteophytic spurs. There being no synovial lining, pain is not present at any point of this process. Physicians should, therefore, not be amazed at the comfort of the laborer with spine stiffened by interlocking hypertrophic spurs, nor should they attempt to attach clinical significance to such changes on x-ray films.

Symptoms arise in hypertrophic arthritis of the *spine* from two sets of circumstances. The commonest perhaps is that in which muscles become spastic from the strain of acting over a changed route from origin to insertion. In chronic form this appears as tenderness on either side of the spinous processes in the cervical or lumbar regions together with paresthetic sensations and a dull ache on fatigue. In acute form it is one cause of torticollis and lumbago. Radicular pains are hardly less frequent, due to pressure on the dorsal root ganglia by bony overgrowths. Thence arise lancinating, darting, stabbing pains. According to their location they may suggest hypertensive encephalopathy, angina pectoris, pleurisy, acute cholecystitis or acute appendicitis, and sciatica. The differential diagnosis must be made by demonstrating the absence of other disease and by the relief rest affords in the cases of arthritis.

Arthritis of the *sacroiliac joint* which is so common in early and middle age, is found rarely in individuals more than sixty. By that time the sacrum and ilium have fused, and the motion of the joint, which must take place if pain is produced, no longer is possible.

There is no need to discuss hypertrophic arthritis in middle-aged persons separately from its manifestations in those of riper years. The degenerative changes are cumulative but variable. Some individuals are old at forty, more at sixty, and all at eighty. The symptoms are about the same if the causative factors are similar. In general, of course, there is less painful arthralgia in old age, because fewer chances for occupational and other traumatizing forces are accepted, and because the sense of pain is duller. Stiffness tends to increase with age. Whether this indicates changes of a fibrous nature in muscles or contractures in capsules and tendons or an unnecessary degree of disuse is not known.

Treatment. Much can be done for the patient by explaining this form of rheumatism to him. The degenerative changes in the joints are permanent

and may be progressive. They are in a sense part of the business of growing old. They must be accepted, with good grace or bad, but they do not impair the general state of health, and they need not lessen activity in any reasonable sense if the limitations which they impose are met. Many people get along with no symptoms at all. Others have minor or transient arthralgias. Only a few have serious difficulty. When symptoms are present, adequate causes are detectable, and simple remedies usually bring relief. Intercurrent diseases, such as respiratory infections, which may be serious for the patient with atrophic arthritis, may be beneficial to one with hypertrophic arthritis, for they do not attack the joints and yet they enforce rest. When the patient understands his situation thus and realizes that he is not to be a cripple, a great load of fear is lifted from him. This may be the only therapy required, or he may become more intelligently effective than ever by experimenting with the adjustments that are necessary in his particular case. Usually after explanation he ceases to be a gullible consumer of rheumatism remedies.

Much can be done to prevent hypertrophic arthritis from becoming burdensome. This is the duty of the general practitioner in his role of family medical adviser. He must supervise carefully the proper setting of fractures and the restoration of normal function. He must watch sprains and disease of the epiphyses. He must shield his patients from occupational hazards as much as possible. He should correct errors of posture when they first appear, not long after they have begun to cause trouble. He should treat obesity as early as he can get his advice accepted, to counteract its mechanical stresses and overcome the eating habits which produce it. To the middle aged and aging he must show how much value there is in the periodic health examination, how it can be used to detect and to minimize the effects of oncoming

Rest is the single essential element in treatment. Stress and strain make the joints hurt. Usually distress has been endured for a long time before relief is sought so that general fatigue is added. Rarely is the arthritis serious enough to require hospitalization or a vacation away from home or work. Eight to ten hours in bed at night in addition to a noon nap may be enough. If possible this should be arranged satisfactorily within the structure of the patient's program. If it cannot, then the program must be altered, for there is no other way of living sensibly with the joints. This is a task which requires all the patience and ingenuity which the physician can command. The middle-aged or elderly housewife, who has been excessively busy at home or abroad with church and clubs and grandchildren, resists strenuously any change of pace or habits. So does her husband, who is facing the crisis of retirement. Even when the benefit of rest has been demonstrated, they may reject it in favor of their accustomed treadmill.

Rest for the affected joints must be *prescribed precisely*. A painful neck can rest only where the patient is lying down without pillows. This position is likely to increase the symptoms at first, for the release of muscle spasm is not smooth. It should be maintained for several periods during the day and night according to tolerance and decreased only as comfort warrants. Middle aged individuals often respond well to a collar, which transfers the weight

of the head directly to the shoulders, or to regular sessions in a suspension device. Old people as a rule do not like them or do poorly in them. Painful shoulders and dorsal kyphosis also require rest in extension. Lumbar lordosis responds to the recumbent position with the thighs elevated or with a small pillow at the small of the back. The bed should have fairly stiff springs and mattress. When sitting down, such patients should select chairs with arm rests and arrange themselves so that their position is both physiologic and comfortable. Hypertrophic arthritic hips require a lying or reclining position. Painful knees may need elastic cloth bandages. Flat feet need good shoes with arch supports or lifts on the medial sides of the heels.

Exercises are very helpful also in overcoming muscle spasms and atrophy and in restoring functional control of the joints. They should not be performed with too much vigor, as in "setting up" drills, nor should they be directed chiefly to "limbering up." The limits of pain must be respected. Preferably the exercises are done in bed two or three times a day after a rest period, and special attention is given to affected joints. Thus, fingers are extended and flexed, elbows extended and arms extended and rotated for affections of the arms, for the hips, gluteal tightening and rotation of the legs; for the knees, flexion and quadriceps setting; for the feet, exercises for the ankles and toes, for the back, stretching and breathing exercises. When up and around, postural exercises are given.

Heat feels grateful on hypertrophic arthritic joints. The simple hot-water bottle or electric heating pad used for one or two periods daily is sufficient. No special benefits are derived from more expensive apparatus. Warm clothing and warm bed covers are especially helpful in the case of old people. Massage is rarely necessary, unless done intelligently, it may cause harm.

The *diet* should be a normal one. Often it has not been in these patients, and improvement in use and comfort seems to follow successful efforts to restore foods which have been omitted through fear or distaste. Milk, meat, vegetables, fruits, and eggs all have their place in an optimal food intake at any age. Meals should be taken regularly and unhurriedly. Obesity in the middle-aged and elderly must be conquered by reduction in the number of calories, not by exercising or thyroid substance. Often it proves wise to allow individuals who have been overweight for a long time to remain somewhat beyond the scheduled ideal for their height and age because they are happier and heartier so.

Acetylsalicylic acid (aspirin) is the best and safest analgesic. It is unnecessary in the majority of cases but can be used in doses of 0.3 to 0.6 gm (5 or 10 grains) to relieve pain which interferes with sleep or proper exercises. *Pharmacotherapy* has nothing else to offer for hypertrophic joints, although much for associated conditions. Vitamin preparations are worthwhile in cases where the diet has been inadequate. The correction of anemia by iron or liver, according to the type of deficiency, is worth while. Thyroid extract should be reserved for those who have definite myxedema, in which cases the symptoms of arthritis respond well. Patients with hypertensive heart disease and mild degrees of congestive heart failure frequently lose minor arthralgias upon return of compensation. Estrogenic substances, given in sufficient doses to control the vasomotor distresses of women in their menopause, often indirectly relieve their joints also. Estrogenic hormones are also

helpful in osteomalacia. Since infection is not concerned with hypertrophic arthritis, penicillin and the sulfonamides are not indicated.

Surgery has much to offer in occasional cases. Traumatic injuries may require operation. Joint "mice" may have to be removed. Fusion of the sacroiliac joint can be induced to stop its discomfort. Various operations have been performed on severe hip disease, designed either to reconstruct and to restore function or to end pain by obliterating the joint. Such matters are best left to conservative orthopedic surgeons. (See Chapter 43.)

PERIARTICULAR FIBROSITIS

There are several ill defined affections of the supporting tissues of the joints which appear to be more or less distinct from inflammatory and degenerative joint disease. They are very common but so poorly understood as to make extended comment unwarranted.

Bursitis. Atrophic arthritis involves the bursae regularly, and gout may do so (see p. 233). There are also instances which seem clearly traumatic in origin, these react in the same manner as hypertrophic arthritis. They show no general or local signs of infection and no interference with health. They appear in acute or chronic forms of the bursae or sclerosed

bursa or a slower accumulation of calcium containing material. The symptoms are those of tenderness directly over the bursa and pain on any motion which disturbs the tissues that outline it. In acute cases the pain may be agonizing. In mild or chronic cases it consists of persistent soreness on use together with aches and paresthesias which radiate along the whole extent of the involved muscles, tendons, and nerve sheaths. Examples are olecranon bursitis in miners, radiohumeral bursitis in tennis players, ischial bursitis in weavers, prepatellar bursitis in housemaids. Adventitious bursae may appear anywhere in response to friction or pressure as over a bunion or over a hypertrophic spur on the os calcis.

In this country bursitis of the *shoulder* is the commonest example. The synovial membrane in that location covers the capsule of the joint and wanders under the deltoid muscle. In chronic forms the capsule is thickened and the tendon of the supraspinatus and the long head of the biceps muscles are frayed. The insertion of the deltoid into the humerus is infiltrated with calcium. Calcium salts also appear in the synovial fluid and may be seen on x-ray films, but the finding has no special significance, for the salts may appear in the absence of symptoms and disappear without treatment. The condition seems to represent about as much hypertrophic arthritis as could be expected. The degree of it is found in every person.

is due to trauma in work or play.

use of the shoulder and tenderness in the region and by demonstrating inability to abduct and externally rotate the arms. Involvement of the joint is excluded by finding that external rotation is free when the elbow is supported. Such acute cases last about six weeks. During this time comfort is possible, if the patient lies flat with the arm moderately abducted. Regular applications of heat and supervised exercises help the pain and shorten the

disability caused by atrophy and spasms. When up, the patient should wear a sling to carry the weight of the arm to the opposite shoulder. Operations to wash out the bursa by puncture or by open reduction are often helpful, as well as x ray therapy. *Chronic shoulder bursitis* is much more common in elderly individuals. In them the pain on use may be much milder or even absent, and there may be no local tenderness. Pain, when present, is likely to be referred to a point near the elbow. There is more atrophy of the deltoid and supraspinatus muscles, more crepitus and limitation of motion on rotation. Such chronic cases require a long course of exercises and frequent periods of rest in a recumbent position to regain function. They should be protected from using their arms too long unsupported as at the piano or sewing. When sitting, their elbows should rest on the chair arms.

Tenosynovitis. This is a less frequent and clinically less disabling affection of the synovial lining of tendon sheaths. It is quite rare in old people.

Fibrositis. In England and Europe this term is used to describe a painful affection of the connective tissues and supporting muscles, distinguished by the palpation of tender *fibroid nodules* under the skin. It is so rare in this country that there is much doubt of its existence. To be sure, muscular aches and pains are part of grippe and many other acute infections and they are found in heat cramps and the "bends," but these are obvious causes and they clear without residue. The acute discomfort experienced when performing unaccustomed exercises wears off also without trace in young people. It takes longer for middle aged and elderly individuals to get themselves into more active states of physical fitness without suffering an aching soreness after rest, but they can do so.

The reasons for this universal "muscular rheumatism" are obscure. Fibroid nodules are found in some, but they are present also in healthy young people, for example over the sacrum, so that they seem unimportant. Fibrous changes in muscles occur in the physically fit as well as in the inactive old person. Perhaps circulatory deficiencies due to peripheral arteriosclerosis or thrombophlebitis are responsible, although it has not been proved that there is less blood flowing through such areas. The theory is attractive, however, because leg cramps seem to be a feature of arteriosclerosis, brought on by exercise and relieved by rest. Also cerebral arteriosclerosis causes fibrillary twitchings, rhythmic, purposeless movements and a rigidity of expression which constitute a type of paralysis agitans. It has seemed to the author that this goes with hypotension more than with hypertension. Workers who have had much experience with pellagra believe that vitamin B deficiency is a factor and report that they can relieve muscular aches and cramps by the administration of the B complex. The author agrees with this in old people on the basis of a few cases. Whatever the causes may be, relief is obtained fairly simply by plenty of heat, i.e., warm clothing, warm rooms, warm baths, reasonable exercise and good food. An aspirin tablet or a glass of wine at night is often cherished by the stiff old man because it warms him up and lets him relax into sleep.

Tight Ilotibial Band. This is a not uncommon cause of backache. If it is shorter on one side than the other, it strains the sacroiliac joint by tilting the pelvis downward at an angle. If both bands are tight, lordosis results.

Dupuytren's Contracture. This contracture of the palmar fascia is seen fairly frequently in old people. Early cases are likely to have an hereditary

basis. Local trauma seems not etiologically responsible, for it is as common in business men as in laborers. The author has been impressed by the fact that all the cases which he has seen in elderly people have shown cervical lordosis and round shoulders. It is always bilateral, the fifth and fourth fingers are most involved, and there is progressive difficulty in extension. The joints and tendons escape, but the skin over them fixes them tightly in flexion. There is no pain. Treatment is unsatisfactory. Surgery fails unless removal of the adhesions between skin, fascia, and tendons is complete.

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CHAPTER 43

DISEASES OF THE BONES

ARTHUR STEINDLER

In no other system in the human body is age reflected by somatic changes more strikingly than it is in the systems of locomotion. The physiologic changes of senescence reduce the resistance of the skeletal system so that it becomes highly susceptible to exogenous influences. Inflammatory factors, traumatic sequelae, circulatory disturbances, and many other pathologic conditions assume a much more severe aspect than they do in patients of earlier years.

PHYSIOLOGY OF SENESCENCE AS IT AFFECTS THE SKELETAL SYSTEM

Senile changes in the skeletal system are characterized by a gradual atrophy of the uncalcified bone as, for instance, in rickets or in the malacic changes. It is rather a natural process of retardation of the osteoblastic activity in the endosteum.

W. Müller¹ calls these changes a fractional necrosis. Although the bone marrow itself is alive, there are areas which show disappearance of bone cells, empty lacunae, and necrotic bone tissue. It seems that a decreased blood supply, due to arteriosclerosis, is behind this bone atrophy. Bone structure is very sensitive to circulatory deficiency, and even more so the marrow. We may expect, therefore, that in people with general circulatory disturbances such findings of molecular necrosis are not unusual. Areas of aseptic necrosis

after traumatism

The circulation is not the only factor in the production of senile atrophy. Defective alimentation, inanition, such as we often see in senile persons, will lead to loss of calcium and phosphorus. Since the excretion of these substances in the urine is maintained, the loss is covered at the expense of the skeletal system. Chemical analysis shows that even where there is a loss of weight, as often occurs in old people, the calcium excretion is maintained and may be even increased. An abundant induction of calcium salts leads to an increased excretion, and if nutrition remains insufficient otherwise, osteoporosis is the result.

One of the most common causes of this condition is inadequate utilization of the ingested calcium, and this again is due to the lowered gastric acid.

In addition there is a decrease of ptyalin in the saliva which accentuates the inadequacy of digestion of the carbohydrates, and which is only partly compensated for by the amylase excreted by the pancreas. The aged also show a relative lack of hydrochloric acid secretion in the stomach and the acidity declines to 30 to 35 after forty years. Likewise there is a diminution in the proteolytic enzymes. It is only natural under these conditions that older people have a lack of appetite, are prone to gastritis, constipation, and impaction (See Chapter 34).

All these factors are reflected in the skeletal system, as a simple bone atrophy which is similar to that seen due to other causes, such as cachexia, malignancies, or advanced tuberculosis of the lungs.

SPECIFIC BONE DISEASES OF THE AGED FROM NUTRITIONAL AND ENDOCRINE CAUSES

Idiopathic Senile Osteoporosis Pathogenesis The *exogenous factors* are deficient dietary regimen, undernourishment and avitaminosis of A and D, and a calcium poor diet. The vitamin content of the blood in the aged is low. The ascorbic acid falls to 0.2 to 0.3 mg. per 100 cc. against a normal of 0.7 to 1.25 mg. per 100 cc. Vitamin C is diminished in the blood serum while lipoids and proteins are increased. There is also decrease of vitamin A and D (See Chapter 12).

Endogenous factors are senile disturbances in the gastrointestinal tract interfering with absorption of calcium, increase of alkalinity or increase of fatty acids may retard or inhibit calcium absorption. There is an habitual hypocalcemia and hypophosphemia in the old, the values ranging from 8 to 10 mg. per 100 cc. for calcium and from 2 to 4 mg. for phosphorus.

Thyroid dysfunction is seen in osteoporosis but is more significant in senile osteomalacia. Parathyroid dysfunction in osteoporosis due to simple senile changes in the parathyroid gland, is also reported. It is only in true parathyroid disease (osteitis fibrosa generalisata), however, that the effect of parathyroid hyperfunction due to adenoma has been definitely established, and that there is an excess of calcium, a lack of phosphorus, and excess of phosphatase (See Chapter 16).

Some see an indication of hypophyseal dysfunction in the fact that osteoporosis in old women may be accompanied by hypertension and obesity. Cushing's syndrome of hypertrichosis, obesity, hypertension and diabetes insipidus is also associated at times with osteoporosis, and this pituitary basophilism associated with adenoma of the anterior pituitary gland reflects the chief sig.

There is

osteoporosis

Furthermore, since all vitamins are necessary for the function of the endocrine glands, a lack of vitamins, peculiar to old age, must in itself lead to glandular dysfunction, and, again, since gastrointestinal dysfunction leads to defective vitamin absorption the former factor must have some share in both vitamin and glandular insufficiency. It appears that it is rather hopeless to reconstruct a successive chain of events from the evidence of insufficiency of the glandular

Pathology. Senile osteoporosis was known and described as a classical entity by Charcot (1863) and Vulpian (1873). It is an eccentric atrophy with coarseness of the internal architecture of bone. The statically important trajectories of the trabeculae (e.g., the vertical lamellae in the vertebrae) are always less affected than the secondary trabecular systems. The atrophy of the cortex proceeds from inside out, yet lacunar absorption is not more extensive than normal and the outlines of the marrow spaces remain smooth. Widening of the Haversian canals occurs. We must assume therefore with Pommer, that the atrophy is due primarily to a restraint in the osteoblastic activity. Other than in the long bones and the spine, the atrophy appears in the maxillary bones and in the skull.

Under physiologic conditions this does not interfere with the normal functions and reactions of bone. Such bone shows the same ability to form



Fig. 149 Photomicrograph showing porotic bone

spurs and bridges as a reaction to static stresses and the same general tendency to the healing of fracture.

It should be noted that it is the lack of bone apposition and not the decalcification or the production of noncalcified bone which is the earmark of the simple senile osteoporosis in contrast with both rachitis and osteomalacia.

This process of senile atrophy easily assumes pathologic proportions under the effect of endocrine, vasomotor or metabolic disturbances. The bones of the aged become more brittle and fragile and more susceptible to fractures and subsequent deformities. Under the influence of body weight, deformities occur in the spinal column further enhanced by degenerative changes in the intervertebral disks. These become disintegrated, often encrusted with calcium and lose their elasticity. The pressure resistance is

low in the marantic osteoporotic bone, hence the tendency to fracture especially in the neck of the femur and in the vertebrae (See Figs 149, 150 151)

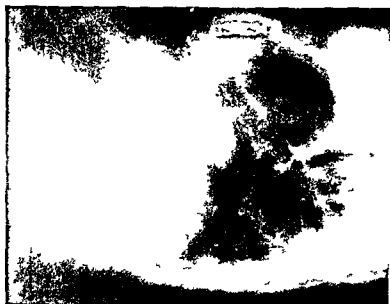


Fig 150 Lateral view of lumbodorsal spine showing fish-tail vertebrae with compressions

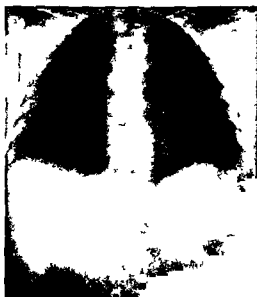


Fig 151 Anteroposterior film of same spine as Fig 150

Clinical Diagnosis Black's² study of 208 cases of osteoporosis at the Mayo Clinic shows an average age of sixty two and a sex ratio, male to female, of 4:1

The spine usually shows a *kyphosis* more often limited to the dorsal spine and its movements are restricted. The head is thrust forward, the thorax is flattened anteroposteriorly, and the sternum is curved and depressed at its base. There is a transverse abdominal crease (Fig. 153).

The patients complain of weakness, fatigue and low back pain which radiates. X-ray films show a decrease in the number of trabeculae, a thinned out cortex, and in the spine tipping and curling out of the edges of the vertebral deformation of the bodies and thinned out disks in the dorsal region.

In the lumbar region where the disks have preserved their elasticity they herniate into the middle of the bodies. The result is a form of biconvex expansion of the disk and a fishtail deformation of the vertebral body. As a result of this vertical flattening there may be a lateral deformity also. While the bones are lacking in calcium there is ample evidence of calcium deposition in other structures: sclerosis of aorta and calcium deposits in the digestive and urinary tracts.



Fig. 157 Typical malacic pelvis

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and cod liver oil 4 cc. three times daily before meals. On the basis of an assumed relationship between basophilic anterior pituitary adenomata and osteoporosis, radiation of the pituitary gland has been carried out with good results and besides vitamins A and D vitamin E has been employed in the after treatment.

To prevent deformity of the spine corsets or better braces are used. We recommend the spring back brace constructed on the Taylor brace.

principle which supports the spine and the anterior abdominal wall leaving the chest free

Local and radiating pain is best controlled by support and by physiotherapy (See Chapter 10) The latter is applied in the form of radiant heat and gentle massage In addition sedatives should be given but with discrimination because of the arteriosclerotic changes the increased capillary permeability and the high blood concentration of the aged Barbiturates



Fig 153 Appearance of patient at entry June 1940



Fig 154 Appearance of patient after four months on low fat and high calcium diet



Fig 155 Same patient April 1942

often show a prolonged effect because of lessened permeability of the kidney Blood transfusions are very useful in raising the general body resistance

That aged patients should be restrained in bed as little as possible to avoid hypostatic pneumonia should be self evident

Case Woman Age 53 years Admitted March 10 1940 Diagnosis Senile osteoporosis

The patient came in complaining of pain in the back of about one year's duration This began when she stretched herself while lying in bed The pain had gradually become worse so that for the past several months she had been bedfast Coughing or sneezing

caused pain. There were no abnormal dietary habits and there was nothing of importance in her past history.

Examination showed a well developed and well nourished white female who was comfortable if she did not move. Examination of head and neck were negative. Heart and lungs were clear, blood pressure 128/90. Motions of back were markedly limited. Motions of extremities were not limited.

The x-ray films showed osteoporosis of spine with wedging. This patient was studied carefully from the standpoint of steatorrhea, parathyroid dysfunction, or other systemic difficulties but the findings were negative.

Treatment: bed rest, spinal support, high calcium, high vitamin diet (see Figs. 149-151).

SPECIFIC BONE DISEASES OF THE AGED FROM HORMONAL CAUSES

Senile Osteomalacia. Senile osteomalacia is merely a type of osteomalacia rather than a separate entity. Pathologists distinguish a juvenile form, identical to late rickets, a puerperal, a climacteric, and a senile form. The essential point is that it is a systemic disease associated with loss of calcium from, and atrophy of, the skeleton.

The *pathogenesis* of osteomalacia is still uncertain, in spite of the many investigations which have been made, especially in the field of ovarian hormones. Experimental studies of Erdheim³ showed that in hyperparathyroidism the healing of fractures is retarded and an osteomalacic callus develops. Hypertrophy of the parathyroid in osteomalacic patients was observed by Erdheim and Schmorl, but whether this hyperplasia is the cause of the condition or is the effect of an increased calcium metabolism is still undecided.

Pathology. The principal feature of senile osteomalacia is the formation of calcium-free osteoid seams in addition to osteoporosis. Especially at points of tension and stress, for instance in the neck of the femur, in the vertebrae, or in the ribs, one sees a calcium-free osteoid substance, finely porotic in structure, which is laid down by way of fibrosis of the marrow and a subsequent osteoid metaplasia of this fibrous tissue. The haversian canals are outlined partly by newly formed osteoid tissue, partly by old decalcified bone. The bone tissue is extremely soft, and often can be cut with a knife. The marrow spaces appear reddish, resembling the pulp of the spleen, and contain an abundance of myelocytes.

Clinical Symptoms and Course. The onset is more or less rapid, as compared with osteoporosis. First symptoms are violent pain in back, flanks, groin, and difficulty in walking. The sternum protrudes (pectus carinatum), the shoulders droop. The trunk is bent forward and there is a deep transverse abdominal crease across the costal margins. There is a marked restriction in spinal motion and the height of the trunk is diminished. Not infrequently there are signs of tetany, such as carpal and pedal spasm, reflexes, clonus, tremor, increased muscular activity, and pupillary signs.

X-ray studies reveal the presence of calcium free bone by the washed-out appearance of the bone and the scant trabeculations, especially in the vertebral bodies and ribs. The dorsal vertebrae appear flattened or of fish-tail shape. In the lumbar spine one sees large biconvex intervertebral disks. The pelvis shows lateral compression with duck-bill shape deformation of the symphysis, although less so than in the osteomalacia of younger individuals. The long bones do not, as a rule, show the marked deformities seen in ordinary generalized osteomalacia. The disease is mostly confined to the spine and thorax.

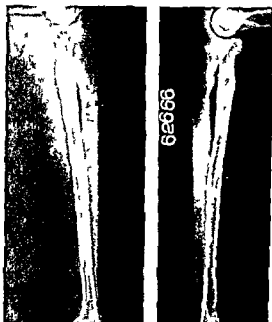


Fig 156 Pseudofracture of radius and ulnae

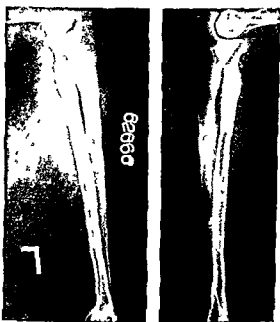


Fig 157 Healing of pseudofractures

The general condition is much impaired by weakness, emaciation, pelvic disturbances, constipation, urinary difficulties, and calculosis. These patients are susceptible to bronchopneumonia because the respiratory motion is impaired.

Differentiation between Senile Osteoporosis and Senile Osteomalacia The skull changes mentioned in osteoporosis are not found in osteomalacia. The

bones in the latter are flexible and do not fracture as easily. In osteoporosis they are hard and brittle and more disposed to spontaneous fracture. Pelvic deformities, so noticeable in osteomalacia, are absent in osteoporosis. Deformity of the spine exists in both conditions, but in senile osteomalacia it is more of the kyphoscoliotic type.

In x rays the osteomalacic bone appears washed out and there is more or less disappearance of bone texture, in osteoporosis there is eccentric atrophy of bone with thinning and atrophy of the trabeculae.

Treatment of Senile Osteomalacia. In the aged the treatment is largely directed toward improvement of the general health and restoration of the calcium phosphorus balance. The serum calcium is usually low and there is a similar reduction in serum phosphorus. Fat soluble vitamin D must be given. For the absorption of calcium and phosphorus more is necessary than the mere administration of vitamin D, however. There must be sufficient gastric acidity and adequate hepatic and pancreatic function to insure the absorption of the fats containing the vitamin D. The administration of bile salts is indicated for that purpose. Beneficial results are obtained with irradiated cod liver oil, the effect of which is said to exceed that of cod liver oil concentrates. Following such therapy considerable amounts of calcium and phosphorus are deposited in the bones as shown by the results of metabolic studies as well as by the increased density of bone seen in x rays.

Calcium salts and parathormone may be used in cases of osteomalacia complicated with tetany, where the calcium content of the blood is especially low. If intestinal disturbances, especially steatorrhea exist, one may give calcium gluconate together with proper dietary measures.

Deformity and radiating pain require immobilization. This, considering the age of the patient, is best carried out in an ambulatory manner by means of corsets or braces, similar to those used in senile osteoporosis.

DEGENERATIVE BONE DISEASE OF UNKNOWN ORIGIN

Paget's Disease. Paget's disease is one of later years, being most common from fort-

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Etiology. There is nothing definite known about the pathogenesis of this condition, although parathyroid disturbances have been repeatedly accused of being responsible.

Pathology. Paget, in his classical description of the bone disease which

bone is most conspicuous in the periosteum of the tibia. The lamellae sur-

marginal layers of the bone marrow, with a loosening of the endosteum followed by catabolism of the lamellae. It is believed⁵ to be a creeping serous inflammation which leads to fibrosis in the marrow and to the formation of new bone in the haversian system laid down in a peculiar mosaic like structure characteristic of the condition. The first changes consist in this loosening of the endosteum then follows absorption of bone then fibrosis of the

periosteum as well as from the endosteum. Mosaic structures begin to appear. The bone becomes thicker. The periosteal fibrous bone becomes mature bone and the endosteal bone apposition produces a narrowing of the medullary spaces.



Fig. 158 Photomicrograph showing typical mosaic structure in Paget's disease

In the second or *intermediate stage* osteoblastic activity holds the balance to the earlier osteoclastic activity. More and more the fibrous bone is replaced by mature Paget bone.

The third is the *healing stage* in which the osteoblastic activity preponderates. The mosaic structures become more marked. The bone becomes more sclerotic, hard and brittle. The fatty marrow is replaced by fibrous marrow (see Fig. 158).

Special Pathologic Features Pseudocysts without lining and brown tumors are occasionally seen in the coarse trabecular pattern.

SKULL CHANGES **OSTEOSCLEROSIS** In the polyostotic form the skull is regularly involved, being hyperostotic, thickened, rough, irregular and having the so-called "nigger wool" appearance in x-ray films. In some cases one also sees definite osteoporotic changes.⁶ These begin with small, rounded

circumscribed areas, usually in the frontal region, which expand in the course of years until the entire calvarium is involved. Often several separate foci develop simultaneously and become confluent. In the majority of these cases typical Paget changes are seen in other bones, but there are cases reported of circumscribed osteoporosis limited to the skull. It seems that the peak of incidence for these osteoporotic skull changes occurs much earlier than those of the usual Paget's disease, indicating a prolonged earlier osteoporotic stage of the disease. The primary disturbance is in the diploic circulation: decalcification and, later, sclerosis follows (See Fig 159).

PAGET'S DISEASE IN THE VERTEBRAE The spinal column is involved in 90 per cent of cases of Paget's disease. The cervical section usually remains free. Most commonly involved are the mid dorsal and the lumbar spine. Here also a malacic or osteoporotic phase occurs preceding that of condensation. Three stages may be distinguished: one of rarefaction and coarse trabeculae

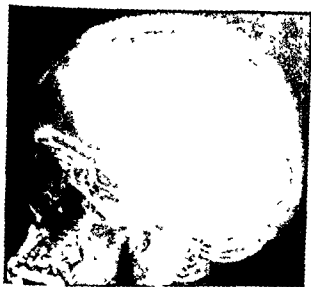


Fig 159 Typical appearance of skull in Paget's disease

tion with vertical trabecular orientation, one of progressive condensation and opacity with deformities, and one of complete condensation with sharp contours. Since these phases may occur simultaneously in different portions of bone, x ray films show rarefaction as well as condensation. There are trabecular changes with abnormal orientation. In newly formed trabeculae, the vertical trajectories which are of static importance preponderate. Later, flattening of the vertebrae, irregular contours, and hypertrophic changes in the processes occur, resembling osteoarthritis.

Clinical Course There are two forms: (1) The monostotic, occurring in one circumscribed area or in one bone, most often in the tibiae, the vertebrae, or the sacrum. This is the more common type. (2) The polyostotic, this involves a number of bones: both tibia, tibiae and vertebrae and, almost always, the skull.

The principal signs are pain, which is dull, boring, osteoconic, and most

common in the tibia, deformity, bowing of the long bones tibia, femur, and secondary static disabilities of the knees The spine assumes a kyphotic deformity, the normal lumbar lordosis is effaced and its mobility is much restricted There is an increased size of the skull due to hypertrophic cranial changes

The laboratory findings include normal calcium, low phosphorus, and increased phosphatase levels in the blood This increase in phosphatase may be very high, it often reaches many times the normal value, up to 100 to 160 Bodansky units Increase of phosphatase is very suggestive of Paget's disease but it is by no means a constant finding, since it depends on the bone activity and hence may disappear in stages of quiescence Besides, there are other



Fig 160 Infiltration of pelvis and hip in Paget's disease

bone conditions, such as osteoporosis renal rickets, osteomalacia, multiple myeloma, and metastatic carcinoma which may be associated with increased blood concentrations of phosphatase during their active stages

Complications **FRACTURES** On the whole, fractures in Paget's disease are rare They are more likely to occur when the bones are large and of extreme density Sometimes they occur both during the active stage and during the healing stage, however Fissure fractures are seen in the cortex on the convex side and may be associated with a small amount of callus, which is usually absent and the callus appears early ⁷

PROTRUDING DISK. Cases have been reported of disk protrusion in Paget's disease of the spine. These have been operated upon with disappearance of symptoms.⁸

SPINAL COMPRESSION. The causes of compression myelitis observed occasionally in Paget's disease are first direct osseous pressure by a collapsed vertebra or some hypertrophic bone formation and second vascular lesions which produce sclerosis or malacia of the medulla. A case of sudden collapse of a Paget vertebra followed by transitory quadriplegia and a persistent Brown Séquard syndrome has been reported⁹ in a patient with most unusual cervical spine involvement. Others have found lesions of the posterior columns alone—simple compression paraplegia, spastic paraplegia with sphincter and sensory disturbances from vertebral compression, progressive medullary compression with block and increased globulin and



Fig. 161 Infiltration of tibia and femur with abnormal bone in Paget's disease

progressive weakness and numbness in both legs with atrophy and absence of reflexes. These cord lesions are due chiefly to narrowing of the spinal canal and occur most frequently in the dorsal spine.

SARCOMA. It is believed that 10 per cent of the cases of polyostotic Paget's disease ultimately show sarcomatous degeneration. There are two types—the fibrosarcoma and the osteogenetic sarcoma. Malignancy seems to occur more frequently at the sites of mechanical stress.¹⁰ Cases have been reported in which preceding trauma seems to have had some connection with the appearance of malignancy. Such a connection could only be admitted, however, if the tumor was known to be absent before the trauma and if the

with Paget like changes in the skeleton and many believe that a sharp division between these two conditions is not justified. Both are said to represent generalized polyostotic forms of osteodystrophy which may have different, although interdependent, causative factors behind them.

From a practical viewpoint we may point to the following features. Paget's disease is never entirely generalized, even in the severest cases. It involves older people and the principal features are hyperostosis and deformities. The pathologic characteristic is the formation of mosaic structures of bone to replace destroyed bone. The destruction begins from the interior of the cortex, then reaches the marrow and the periosteum. The blood calcium level is normal, but the phosphatase is frequently high.

Von Recklinghausen's disease involves younger people, there is universal involvement of the skeleton, brown tumors may be seen in the porous, malacic bone. There is more tendency to fractures and more general pain, there are no mosaic structures. The destruction begins from the marrow cavity. There is hypercalcemia, hypophosphatemia, and, lastly, there is the parathyroid tumor.^{3, 11}

In spite of these differences there are numerous confusing case reports. Some show severe skeletal decalcification with typical Paget's changes in the skull, a normal blood calcium, mosaic structures, but parathyroid adenoma.¹²

Do such tumors exist in Paget's disease, or are we dealing with hyperparathyroidism simulating or associated with Paget's disease? Gutman and Parsons¹³ report such cases where hyperparathyroidism simulated Paget's disease because of atypical localized hyperostosis and high blood phosphatase, but with hypercalcemia and a parathyroid tumor, the removal of which brought relief, or with enlargement of the skull, but normal phosphatase levels, general decalcification, cysts, hypercalcemia, and a parathyroid tumor, the latter, likewise, being removed with subsequent cessation of symptoms. True combinations of Paget's with von Recklinghausen's disease also seem to have been observed, e.g., a case of bowing of the tibia, thickening of cortex, coarse trabeculation, low phosphorus, high phosphatase in the blood, but with hypercalcemia and a parathyroid tumor. Removal of the tumor gave an initial improvement of symptoms, and the calcium returned to normal. The pain reappeared, however, and the high phosphatase of the blood remained.

Treatment. There is no treatment known which has any effect upon the disease as such. It may become arrested spontaneously. One may obtain symptomatic relief of pain, however.

Iodides, arsenic, and endocrine therapy have been tried, but the effects are doubtful. Lately, *aluminum acetate* (al acetate $\frac{1}{4}$ oz., syrup 300 minims, essence of cherry 4 minims, mel depuratum ad 4 oz., minimum dose 1 drachm, four times daily after meals, combined with 1 pint of milk daily) has been advocated on the supposition that it reduces absorption of phosphorus. Helfet¹⁴ reports eight cases so treated with reduction of pain, diminution of fatigue and gain in weight.

Clinical improvement is further reported after *parathyroidectomy*,¹⁵ based on the idea that the parathyroid glands play a direct role in Paget's disease. It is believed that both the latter and von Recklinghausen's disease develop as a result of a continuous but ill understood intoxication.

The *x ray treatment* for pain, likewise, gives temporary relief only. It

is worth trying before one resorts to surgical intervention. Ultraviolet rays have given encouraging results in some cases.¹⁶ Heat in one form or another sometimes gives momentary relief, otherwise its value is very doubtful.

Surgical treatment for pain by periosteal stripping has been advocated. Drilling or windowing of the bone, with relief of intraosseous pressure, alleviates pain, as our personal experience has shown in some cases. The relief is temporary, but may last for a number of months to a year.

In spinal compression with paraplegia or spasticity decompression (laminectomy) has been carried out with success. Schwartz and Robock¹⁷ report nine cases of their own and an additional ten from the literature which

cord, probably due to narrowing of

Of three patients operated upon by

operated cases from the literature, one patient died and four recovered from the paraplegia.

Case White male Age 53 years Admitted April 19, 1933

The patient came in complaining of weakness in the right knee since 1931. He gave a history of fracture of right femur when he fell from a tree sixteen years ago but until two years ago he was quite well and worked every day. The right thigh and lower portion of his back began to ache gradually during the past two years.

Physical examination showed a fairly well developed white male. There was $\frac{1}{2}$ inch shortening of the right leg. There was slight equinus of the right foot. The right knee was in slight genu varum and there was some increase in temperature of both right tibia and femur. There was considerable limitation of rotation in both hips. The patient stated that his head had been enlarging. He wore a size 8 hat.

Heart and lungs normal. No edema. No abnormal reflexes. No Babinski.

X with braces

NEOPLASTIC BONE DISEASES OF THE AGED

Multiple Myeloma. *Statistics.* The age range of multiple myeloma is thirty to seventy five, with the peak of incidence at fifty five years. It is believed that multiple myeloma spreads from bone to bone and does not involve internal organs. Metastases to internal organs are not rare, however, and have been found in the spleen, liver, and lymph nodes more rarely in the tonsils, thyroids, suprarenals, ovaries, and meninges. The hematopoietic tissues are preferred and pulmonary metastases have not been found. In rare instances, the soft tissue metastases may even give rise to the first clinical manifestations, for instance, metastases in the cervical lymph glands may simulate Hodgkin's disease.

Pathology. The most common location of multiple myeloma is in the skeleton, particularly in the pelvis and the shoulder girdle.

These tumors consist of bone destructive, very vascular, round tumors of the consistency of clotted blood and of a purulent or hemorrhagic color. Microscopically these tumors are composed of dense sheets of cells (plasmoid cells) with eccentric nuclei, spoke like arrangement of chromatin, and with an acidophilic cytoplasm. In size they vary from small cells with dense nuclei to larger cells resembling plasma cells or myelocytes. According to the cell type, we

speak of a lymphocytoma, plasmacytoma, or myelocytoma, but they all seem to be different stages of a common single type. Their origin is a marrow



Fig 162 Photomicrograph of typical field of multiple myeloma

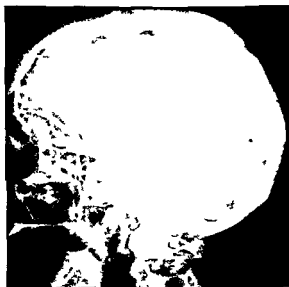


Fig 163 Punched-out areas in skull in multiple myeloma

element and not the blood. The white cell count of the circulating blood is not increased greatly (see Fig 162).

In x ray films the lesions appear as multiple areas confined to the location

of the red marrow and typically central in the bone. They are round, sharp areas of translucency and cannot be distinguished from metastatic carcinoma except that myeloma does not usually show lung metastases and carcinoma appears more often as a single lesion. These multiple punched-out areas sometimes become more diffuse by confluence and give a mottled, honey-combed picture, especially in the ribs. The skull likewise shows multiple isolated punched-out areas, particularly in the frontal and parietal regions (see Fig. 163).

Clinical Diagnosis The symptoms are ushered in by general malaise, increasing weakness, anemia, weight loss, and by intermittent rheumatic



Fig. 164 Punched out areas in spine and scapulae in multiple myeloma

pains, especially in the lumbar region. Girdle sensations are often complained of in the lower thorax. Later there is continuous pain, increasing deformity, kyphosis, swelling, multiple fractures and, finally, severe cachexia. Skeletal deformities appear principally in the spine in the form of dorsal kyphosis, sunken sternum, and as deformities following pathologic fractures.

The blood picture shows a moderate leukocytosis, 11,000 to 15,000, with 1 to 10 per cent myelocytes and myeloma cells in the blood stream. In a number of cases there is an increase in the concentration of plasma protein, up to 14 gm per 100 cc (as against 6 to 7 gm per 100 cc normally). A secondary anemia of two to three million red cells is common.

Bence Jones' albuminous bodies are found in the urine in 65 per cent of the cases, but if present they are not pathognomonic, since they are also found in metastatic carcinoma and in the leukemias. Calcium and phosphorus concentrations in the blood are normal, phosphatase is occasionally increased.

Complications **FRACTURES** Pathologic fractures occur in the ribs, the spine, and the long bones. They are particularly common in the ribs, being found there in 63 per cent of the cases. The area around the fracture appears rarefied, and while deformities are marked they do not have bends or twists seen in osteomalacia or in von Recklinghausen's disease.

VISCERAL There are no pulmonary metastases, but secondary pulmonary complications, such as bronchitis and emphysema, are common.

NEUROLOGIC The most frequent complications of myelomatosis of the vertebrae are root symptoms and cord compression.¹⁸ Root symptoms are caused by direct pressure, cord lesion and paralysis by the pressure of the fractured or collapsed vertebrae.¹⁹ The thoracic spine is involved most commonly. The outstanding clinical signs are local vertebral tenderness, severe radiating pain, sometimes but not always relieved by recumbency, and herpes. Brachial neuritis, due to root compression, is occasionally observed. Signs of cord compression are paresis, paralysis, sensory disturbance, and spasticity of lower extremities.

Prognosis The prognosis of multiple myeloma is poor. The average duration of life is two years. Remissions are sometimes observed.

Treatment Practically all the treatment is symptomatic. No proven cure has been observed. Palliative treatment for the relief of pain consists of anodynes and x ray therapy. For the support of the general condition, high calcium and vitamin D diet is recommended. Intractable pain may be relieved by high cordotomy. Coley has claimed an arrest of three to five years under his treatment with mixed toxin of *B. prodigiosus* and streptococcus.

Lately a new remedy has been introduced into the treatment of multiple myeloma which holds out some prospect of arresting the disease.²⁰ The drugs stilbamidine and pentamidine are two diamidine compounds which were used by Snapper on multiple myeloma, in which an increase of the globulin content of the serum is a frequent occurrence. The first injection is 50 mg. followed two days later by 100 mg. and thereafter 150 mg. daily. Fifteen patients were so treated and all of the patients were kept on a diet low in animal protein. A considerable improvement was obtained. Snapper believes that these substances have a specific influence on the disease since there is a rapid subsidence of pain. The disease is not cured, but is only temporarily checked. The author warns, however, against premature ambulation because the skeleton is still intensely involved and immature mobilization may lead to fracture. A series of fifteen or more intravenous injections of these drugs combined with a diet low in animal protein relieves pain in most cases of multiple myeloma. Relapses occur. Eleven of his patients were able to walk when they were discharged from the hospital.

Case White male Age 50 years Admitted May 13, 1933

. . . 1972 with

in the left chest, much more acute than the pain in the back. The pain seemed to . . .

increased since 1931. In December of 1932 the patient noticed blood in the urine; this lasted for two days and was not noticed again. Four months before admission he coughed some bloody sputum on three occasions. The cough continued, but the bloody sputum stopped. No familial history. Patient believed he had lost 40 pounds within the last year.

Physical examination was negative, except for tenderness along the course of practically all ribs.

Laboratory data. Urine was negative except for Bence Jones protein. Hb., 64 per cent, RBC 2,720,000, WBC 6900. Wassermann negative. Blood chemistry: blood cholesterol 216, calcium 13.4, phosphorus 2.86. X-ray films negative except for arthritic changes. May 22, 1933, biopsy of rib on left side: multiple myeloma (plasma cell type). (See Fig. 163.)

Metastatic Carcinoma in the Aged. Carcinomata which are most likely to produce bone metastases are those primary in the prostate, thyroid, breast, and kidney. The general frequency of bone metastases was established by Geschickter and Maseritz²¹ in 334 cases, among which the primary seat could be found in 297: in 134 the metastases were from the prostate, in 100 from the breast, in 22 from the suprarenals, in 7 from the uterus and the ovaries, in 6 from the thyroid, in 10 from the gastrointestinal tract, in 5 from the skin, in 4 from the lungs, in 3 biliary, and in 6 others were combined metastases. Small, slow growing cancers metastasize more readily in bone. The metastases may be either of the osteoblastic or the osteoclastic type. The age range for carcinomatous bone metastases is from forty to eighty years, with the highest incidence at fifty-five years.

The bones most affected are spine, pelvis, femur, and ribs. Almost 50 per cent are single lesions, usually situated at the end of the long bones at the point of entrance of the nutrient artery. An equal number produces diffuse, multiple lesions, especially in the spine, pelvis, skull, and at the upper end of the long bones, near the pelvis or shoulder girdle.

Pathology. The tumor produces a central area of destruction in bone, without expansion of the cortex. With few exceptions (prostate), the metastasis is osteolytic and its pathology varies with the primary tumor.

X-ray Appearance. The principal feature comprises the etched-out areas in the medulla, sharply outlined and not distinguishable from multiple myeloma. The latter, however, is more often multiple, more frequent in ribs and clavicle, and there are no pulmonary metastases. In the spine the vertebrae may be so intensely involved as to show massive collapse, yet the intervertebral disks are preserved, contrary to what one sees in inflammatory destructions. Cysts differ from metastatic carcinoma in that they show signs of ossification in the bone shell and thickening in the areas surrounding the walled off cyst. The osteolytic sarcoma has similar bone destruction but it is more asymmetrical: there is early perforation, and periosteal reaction (Codman's triangle). Paget's disease, in the osteoporotic stage, is often multiple in the tibia, skull, and pelvis, but shows marked periosteal activity and deformities not seen in metastatic carcinoma.

Special Features of Carcinomatous Bone Metastases Coming from Various Primary Sources. FROM THE PROSTATE. The incidence of skeletal metastases from carcinoma of the prostate is reported to range from 13 per cent^{22, 23} to 70 per cent.²⁴ The average age of the patient is sixty-four years. The interval between recognition of the primary tumor and the finding of bone metastasis is stated to be 22.8 months, and the expectancy of life averages one to two years. In 92 per cent of the cases the site of metastasis is the pelvis and the spine. The tumor is osteoblastic. If the prostate is small, the

original tumor may easily be overlooked. In bone metastases, tumor cells enclosed in the bone assume the function of osteoclasts. Extensive resorption by tumor osteoclasts occurs, but the invasive power of the tumor is low, so that proliferation of bone by tumor osteoblasts or connective tissue osteoblasts prevails (see Fig 165). Pathologic fractures occur only in 2 per cent of the cases.

Case White male Age 62 years Admitted May, 1937

Patient fell and injured his right hip about two months before entry. He stayed in bed about two weeks then he got up but developed shooting pain down the back of the left thigh to the leg. Patient gave a history of some difficulty in starting urinary stream.

Physical Examination Emaciated male in acute pain in region of left hip. Heart and lungs were clear. Blood pressure 145/60. Motions in all joints of extremities were within



Fig 165 Photomicrograph showing tumor cell infiltration with new bone formation

normal limits for a man of his age. Motions in spine were limited. Prostate was enlarged but not stony hard.

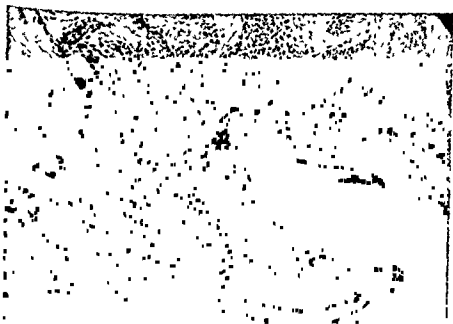
Laboratory Data Tuberculin and Wassermann reactions negative. Blood count WBC 5000 RBC 3 500 000 phosphatase 142.5 (Je prostate, resected tiss)

Treatment of metastases of carcinoma of the prostate with stilbestrol has been found to exert a remarkable influence upon skeletal metastases. The effect is largely subjective, consisting in control of pain. The x ray picture also shows in many instances resolution of carcinomatous metastases in the pelvis and spine and other portions of the skeleton.

FROM THE BREAST The incidence of bone metastasis in carcinoma of the breast is 20 to 25 per cent, the age range being thirty five to fifty five years. Pelvis, spine, femur, skull, and ribs are involved by preference and in one third of the cases the metastatic lesion is solitary. The average time interval



Fig. 166 Infiltration of pelvis and head and neck of femur by osteoplastic tumor from prostate gland



between the first clinical evidence of the primary tumor and that of the metastasis is 39.2 months. The size of the primary tumor bears no relation to the likelihood of metastases.

Symptoms of breast metastases in the spine are rheumatoid and abdominal pain, sciatica, numbness, weakness and paralysis. Pathologic fractures occur in 15 per cent, as against 62 per cent in multiple myeloma, 45 per cent in cysts, 14 per cent in giant cell tumors, 8 per cent in osteogenic sarcoma, 5 per cent in Ewing's tumor.

In the more common osteolytic type there is destruction of spongy and cortical bone by direct contact with tumor cells and, to a lesser degree, by genuine osteoclasts. The spread is by way of the medullary cavity and the haversian canals. A reaction takes place by transition of the fibroblasts into osteoblasts which, by the formation of true bone, protect against further invasion of the tumor, especially in irradiated cases (see Fig. 167).

Case White female Age 50 years Admitted November 10 1941

Patient came to the University of Iowa Hospital complaining of pain in the right hip and loss of weight for the past four months. She gave a history of radical mastectomy in October 1940. She had an uneventful operative course and returned to work in several months. In April 1941, she began to have vague pains in the region of the right hip and knee. By August the pain had become so severe that the patient became bedridden. She had lost about 50 pounds.

Physical examination showed an undernourished white female of about fifty years. Heart and lungs were clear. Blood count normal. No bone pain. X rays given x rays.

A treatment similar to that by stilbestrol for carcinomatous metastasis of the prostate has been recently applied to metastasis from carcinoma of the breast. The hormone in this case is the male hormone, testosterone. It is applied in 50 to 100 mg. doses intramuscularly, daily, especially in metastasis involving the spinal column. The effect is similar to that of stilbestrol in that it improves the general condition and well being of the patient, controls the pain to a great extent, and in general gives the patient a remarkable degree of comfort.

It produces secondary male sex characteristics like increased growth of hair on the upper lip or over the abdomen and the voice often changes to a much deeper scale. We have applied this method in fourteen cases and with few exceptions there was a remarkable subjective improvement noted following the application of this drug.

FROM HYPERNEPHROMA Metastasis into bone frequently follows traumatism. As a rule the metastatic growth is rapid, but cases have remained stationary under radiation treatment. The age range for hypernephroma is from twenty one to eighty-one years. Metastases develop in the old as well as in the young with the peak of incidence in the sixth decade. The bones principally affected are in order humerus, spine, femur, pelvis, ribs, bones of the foot, skull, and sternum. The metastatic tumors are of brownish yellow color. There is marked destruction of the cortex, diffuse involvement of the medullary cavity and of cancellous bone. The cartilage is not affected. Metastatic pulsating tumors have been reported occurring at the distal end of the forearm.²⁵

Microscopically, the tumor shows the typical structure of a hypernephroma. The cells are granular or foamy, with a clear cytoplasm. There is no tendency to walling off. In x rays the tumors appear as punched out defects.

with no periosteal reaction. In contrast to the skeletal metastases from the breast those from a hypernephroma occur more often as single lesions and are more osteolytic.



Fig. 168 Infiltration of hips and pelvis by osteoplastic tumor from carcinoma of the breast



Fig. 169 Osteoclastic metastases to the humerus from carcinoma of the breast

The bone lesion may be the first indication of a primary tumor. Usually there is swelling, rheumatic pain, and anemia. Pathologic fractures occur in 45 per cent of the cases.

FROM THE THYROID Cancer of the thyroid itself is rare, but bone metastases from such cancers are relatively frequent. Ehrhardt reports sixty-six

skeletal metastases in 238 cases, in frequency they range behind tumors of the breast and prostate only (Copeland and Wegelin, 40 per cent)

The primary thyroid tumor may be entirely latent Multiple bone metastases occur in the femur, pelvis, and skull The lesions usually appear near the epiphysis as central or periosteal areas Cases of benign goiter have been reported to produce malignant metastases There is marked and rapid proliferation of tumor cells

The age ranges from forty years on There are progressive emaciation, anemia, local pain, and tenderness

FROM THE BLADDER Metastases to bone are rare The Cancer Registry of the American Urologic Society gives an 8 per cent incidence of metastases Livingston²⁶ reports twelve cases from the literature, six osteoclastic, four osteoblastic and two mixed The cells are of a transitional type resembling those of cancer of the bladder The squamous cell carcinoma has a greater tendency to metastasize than the papillary Metastases develop late in the disease The usual sites are ribs, vertebrae, and pelvis

FROM THE UTERUS Here also incidence of bone metastases is low, 1 to 5 per cent²³ The average age is forty-seven years The tumor is of the destructive osteoclastic type, with no tendency to ossification Symptoms are pain and swelling of the extremities and involvement of the regional glands While cancer of the uterine body is rare, it invades the lymph nodes and therefore remains localized to the uterus and its vicinity Cervical carcinoma invades the parametrium early and involves the surrounding organs The locations of the metastases are pelvis, femur, humerus, and skull

FROM THE GASTROINTESTINAL TRACT Bone metastases from gastric carcinoma are rare Kerr and Berger²⁷ report 2.4 per cent bone lesions in 123 cases They tend to occur at the sites of red bone marrow spine, ribs, femur, sternum, and pelvis Site, size, and type of the original tumor seem to have little influence upon the metastases They disseminate through the blood stream and are osteoclastic, osteoblastic, or mixed in character The bone involvement from cancer of the esophagus is still less common, being only 1 per cent according to Geschickter²¹

Metastases from the liver to bone are likewise rare Up to 1937, only eight cases had been reported in the literature The incidence is given at 2 per cent Metastases in the sacroiliac joint are reported²⁸

Probably the highest incidence of bone metastases from the intestinal tract is found in cancer of the rectum In the coccyx it is given at 5 per cent, while in other bones it is only from 0.5 to 1 per cent

FROM THE LUNGS Bone metastases from cancer of the lungs occur more frequently than is generally assumed, although cancer of the lung itself is rare Skeletal metastases from primary lung tumors are rated variously from 29 per cent²⁹ to 16 per cent²² The more frequent locations are femur, spine, ribs and skull, clavicle, and sternum The average age of the patients is thirty nine to fifty five years One case is reported metastasizing in the superior maxillary bone³⁰ Primary pulmonary carcinoma is a destructive, osteoclastic tumor showing only slight attempts at protective fibro-ostosis in the osseous metastases

Points in the Differential Diagnosis of Carcinomatous Bone Metastases
SARCOMA Most sarcomata are osteolytic They have a tendency to asymmetrical growth, are rapidly destructive, and break through the cortex and

penosteum readily. The age and history are of paramount diagnostic importance

MULTIPLE MYELOMA X-ray findings are almost identical but the lesions are eminently multiple. There are no pulmonary metastases. Bence Jones' protein is present in the urine in 65 per cent of the cases.

CYSTS The symptoms appear late, many remain asymptomatic. There are signs of ossification in the bone shell. The sites are the metaphyses of bone. The patients are children and young adults.

PAGET'S DISEASE It is especially important to distinguish Paget's disease from metastatic carcinoma of the prostate. The phosphatase content of the blood is increased in Paget's disease and may also be increased in metastatic prostate carcinoma of the osteoblastic type. It has been found, however,³¹ that the normal prostate is rich in phosphatase with optimum activity at a low pH of 5 (acid phosphatase), while in Paget's disease the phosphatase activity is not increased at the pH level, rather, it reaches its greater values at a higher pH index (alkaline phosphatase).

TREATMENT OF CARCINOMATOUS METASTASES IN BONE The treatment of bone metastasis is symptomatic and consists largely in the control of pain and improvement of the general condition. Special drugs such as stilbestrol and testosterone for treatment of carcinomatous metastasis of prostate and breast have been mentioned above as has also the stilbamidine treatment for multiple myeloma.

X RAY TREATMENT In breast metastases the value of roentgen therapy has been emphasized. The average duration of life, after bone metastases have appeared, is given as follows by Geschickter and Copeland²³ (1) Cases with radical breast operation and skeletal metastases, average duration of life with irradiation (1000 r over each lesion), 18 months, radical breast operation and skeletal metastases, not irradiated, 11.6 months (2) Simple amputation or excision, with skeletal metastases, irradiated, 16.5 months, not irradiated, 12.8 months (3) Inoperable cases with skeletal metastases, irradiated, 10 months, not irradiated, 7.8 months.

As to the palliative effect of x ray treatment on pain from skeletal metastases, Dresser³² gives the following figures. Group I, less than forty-five years old, definite regression of tumors, relief of pain, general improvement, 40 per cent, relief of pain, no regression of tumors, 43 per cent, no response, 17 per cent. Group II, postmenopausal, no regression of tumor, but relief of pain, 48 per cent, no response to x-ray treatment, 42 per cent.

The bone metastases from hypernephroma are relatively radio resistant. The x ray dosage is the same as in breast metastases, namely, 1000 r per target. Some control of pain and some prolongation of life are obtained. On the whole, irradiation alone offers as much as surgery plus irradiation.

In instances of metastasizing uterine tumors irradiation also offers some relief of pain, but the result is more transient than in cases of metastasizing breast tumor. The same may be said of bone cancers from the thyroid, which are all fatal within two years.

CHORDOTOMY This procedure, suggested by Spiller in 1907, was first used to relieve pain in malignancies of the sacral area. It has now a wide application in controlling pain from metastasis in the entire spine. It consists essentially in cutting the pain-conducting fibers of the anterolateral columns, between the anterior and posterior roots, carefully avoiding any injury to the lateral pyram-

idal tracts. It is, of course, a measure of last resort and should not be undertaken until all other methods to control pain have failed. Frequently the op-

tive of pain control is concerned.

SUPPORTIVE TREATMENT This consists of a well balanced, high caloric diet devised with due regard to the protracted recumbency to which the patient is often subjected. Tonics and blood transfusions are useful in cases of anorexia and anemia. Proper elimination should be maintained by enemas rather than cathartics. Ample fluids should be given. Emaciation and cachexia always appear in the course of the disease, but they often appear very late. At times it is remarkable how long the appetite of the patient will be maintained and his general condition remain fairly satisfactory, except in cases where the gastrointestinal tract is involved.

The greatest comfort can be given to the patient by proper nursing. Pains-taking care of the skin, avoidance of bed sores, regular turning and bathing of the patient and, in incontinence, which is not infrequent in the later phases of the illness, painstaking cleanliness will contribute greatly to making the patient comfortable during his incurable illness.

FRACTURES IN THE AGED

In advanced age bone becomes fragile, less elastic, and more exposed to fracture due to the preponderance of absorption over the osteogenetic activities in senile osteoporosis. After thirty years of age, the ratio between diameter of medullary canal and thickness of compact bone is increased in the femur and humerus, and x-ray films show rarefaction of the spongy bone and enlargement of the haversian canals.

The predisposition to fractures is based on the loss of unit strength, due to this eccentric atrophy of the long bones.³³ It was once thought that these bones fracture more easily because they are more brittle. This is not true, however. In the newborn, compact bone has 50 per cent organic material, in the adult, 40 per cent, in older bone it actually increases to 42 per cent. Therefore, the cause of fragility is not due to less organic content but to loss in thickness of the bony wall and destruction of the lamellar systems.

Because of the senescent changes in bone, repair cannot be normal. There is delayed union and, frequently, nonunion. According to Pommer, these changes are due to arteriosclerosis of the vessels of the bones. Naturally, other factors are also active, particularly those which have to do with decreased calcium fixation and the numerical reduction and lipoid degeneration of the bone cells themselves. It seems that in the aged the phenomenon of bone apposition and functional adaptation, which is a sign of normal life of bone, is greatly retarded and diminished. We have already mentioned certain effects of these changes on the gross morphology of bone, diminution in size, and certain deformations, especially in the spine. Where these occur, a senile curvature develops, enhanced by the general atrophy of bone and certain degenerative changes in the intervertebral disks.

Similar degenerative changes occur in the joint cartilage. albuminoid degeneration, calcification, and ossification. The calcification of cartilage is part of a general process of calcification of senile tissue. (See Chapter 42.)

While the cartilage changes play a great part in the development of articular deformations, especially in osteoarthritis, the bone changes largely determine the frequency, site, and healing properties of senile fractures.

Fracture of the Neck of the Femur. These are common in the elderly and must be considered both from the viewpoint of the patient's general physical condition and as a local problem. Can the patient remain recumbent? Can he stand immobilization? Can he stand skeletal traction? Can he stand operation? Can he stand anesthesia?

Patients who are very feeble and fragile are exceptional. In these, fracture is sometimes a terminal event. The complications of hypostatic pneumonia and uremia must be taken into account. Shock alone may be sufficient to cause death. It is obvious that great concessions must be made in the treatment. These patients will not tolerate

tension

their bed

side, and should be made to sit up in bed at the earliest possible moment to prevent hypostatic pneumonia and bed sores. The majority of the aged suffering from fractures of the neck of the femur or from intertrochanteric fracture are in condition to stand a minimum of surgical interference, however.

From the viewpoint of the local condition, the questions which were most perplexing in former years have now become much simplified. The method of internal fixation of fractures of the neck of the femur (pins, screws, nails) and the Whitman-Leadbetter method of reduction are invaluable because (1) reduction, which is the first prerequisite of union, is obtained by these methods in a large percentage of cases, and (2) following reduction, retention by means of metallic pin or nail fixation does not require plaster immobilization or long bed rest. Patients are able to leave the bed on crutches in one to two weeks. Weight bearing must be avoided for six months by the use of a caliper splint. This removes the greatest objection to cast treatment in old patients. It avoids hypostatic complications and thrombosis, and makes early home treatment possible. It is found by most observers that advanced age is no particular obstacle to treatment. Patients in the nineties are often cited as examples.

The question of treatment becomes more difficult in cases of nonunion following fracture of the neck of the femur, because the operative procedures are not so simple as the mere subcutaneous insertion of nail, pin, or screw. The difficulties so often met here in the aged are excessive deformity, which makes alignment impossible, a necrotic or extremely atrophic femur head,

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required (see fig. 110)

Fractures of the Spine. In evaluating the fractures of the spine we will consider not only the great reduction in the osteoporosis of the vertebral bodies but also the

While these structures lose nothing in their consistency, in fact become harder and more fibrotic with age, their elasticity as buffer joints is greatly diminished and therefore they are unable to sustain shocks and bending stresses.

That the intervertebral disks degenerate comparatively early in life is well known. It seems that these changes set in as early as the third decade. At this time there are noted the beginnings of wear and tear and of fibrillation of the cartilage. By the fifth decade the cartilage already shows numerous protrusions of the disk into defects in the cartilage plate and by the sixth and seventh decades there is the greatest amount of degeneration to be found, such as thinning of the disk and protrusions into the body of vertebrae as so-called Schmorl bodies. The nucleus pulposus shows complete loss of structure.

It is no wonder, therefore, that the disk has practically lost its function as a buffer joint. In response to this, Nature tries to obliterate intervertebral movement altogether by the production of spurs and bridges between vertebrae. Willis³⁴ pointed out long ago that the osteoarthritis of the lumbar spine



Fig. 170 Fracture of neck of femur, illustrating the Lorenz type of operative minimum.

is almost a physiologic process in step with the rapidly increasing degeneration of the disk. In certain types of anatomic build³⁵ we find that these spurs and bridges over the vertebral disk occur regularly after the fiftieth year, as do also

of the degeneration of the intervertebral disk.

We have in mind, especially, the simple compression fractures from minor injuries which are due principally to the greatly increased fragility of the vertebral bodies. There is little or no displacement and no injury to the cord. These fractures, occurring after small jerks and jolts, are so frequent in the aged that any complaint of back pain, especially in the lumbodorsal region, appearing after any kind of accident should be carefully checked by x ray study (see Fig. 171).

How can this type of fracture of the spine be treated with the least

amount of recumbency and inconvenience? Fortunately, these cases are easily controlled by light support (we use a so-called spring back brace) and physical therapy (see Fig. 172)



Fig 171 Compression of lumbar vertebrae forming the so-called fishtail vertebrae

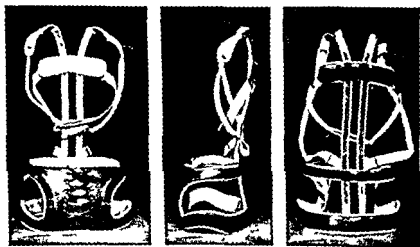


Fig 172 Spring back brace used by the author

stricter sense of the word but rather an acute absorption of the bone on a neurovascular basis revealed by a mottled appearance in the x ray film. It is not uncommon in the aged after fractures or other injuries of the lower fore arm and the ankle and of small bones of wrist and tarsus. It is a reflex neurotrophic phenomenon and while it is not confined to old age it is seen much more frequently in people beyond fifty years (see Fig. 173).

In Colles' fracture such atrophy is by no means commensurate with the deformity. We see well aligned fractures with marked atrophy and functional disability while others with marked deformity may show comparatively little



Fig. 173 Typical spotty atrophy of Sudeck's type of lesions

functional impairment. Rather it seems that the circulatory condition of the soft tissues subsequent to fracture is the important factor. For this reason it is particularly essential to provide for early motion and improvement of the circulation of the soft structures by physical therapeutic means (See Chapter 10).

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SECTION IX

DISORDERS OF THE CUTANEOUS SYSTEM

CHAPTER 44

DISEASES OF THE SKIN

HERBERT RATTNER

THE first visible manifestations of approaching senescence are usually seen in the skin, for the skin reflects practically all of the pathologic as well as the normal changes that occur in the body. Although cutaneous changes appear insidiously and do not affect all persons alike, still the face of an individual is a fairly true index of his years. The changes brought about by advancing years are in the turgor, color, thickness, smoothness, and contour of the skin, involutional changes all, which result from the fact that as a normal process the specialized cells of the skin, rather than its connective tissue framework, are affected by age (See p. 68.)

Normal Aging. The extent, the rate of development, and the particular age at which skin changes become manifest are largely a matter of family trait, as for instance, graying of hair and wrinkling. Whatever the traits, the conversions are inescapable and eventually the skin becomes inelastic like parchment, loses its velvety thickness, and wrinkles develop from degeneration of the elastic tissue, muscle fibers, and subcutaneous fat. With age, the skin gradually becomes more flaccid and less elastic and later thin, dry, and harsh in texture. As the atrophic process progresses, the skin assumes a glossy appearance, the underlying blood vessels become visible, and the color is tinged with a muddy yellow. These transformations are all of an involutional character and are followed by the appearance of degenerative changes, of which the first to develop are the pigmented spots so frequently observed on the face and backs of the hands. These, the so-called senile spots or senile freckles, are frequently the beginning of more ominous senile keratoses. Mingled with them may be tiny atrophic white spots and small clusters of dilated blood vessels, the bright red senile angiomas.

Appendages of the Skin. These also undergo modifications in old age. The changes which take place in the hair are a striking, if not reliable, sign of advancing years. The tendency for the hair to whiten or to thin out even unto complete baldness, particularly in men, is usually associated with old age, although it may occur prematurely. Conversely, it is known that the members of some families retain their hair in its natural state and color into extreme old age. As age progresses the lanugo hairs become thinner and less vigorous, while the hairs of the nostrils and ears in contrast grow longer and stiffer, much like bristles, often the eyebrows become bushy. In elderly women the hairs on the upper lip and scattered hairs on the chin increase in thickness and

length. Similarly hairs in moles tend to grow longer, and the moles themselves become more pigmented (See p 63.)

With increasing age the nails become harder, thicker, and darker in color, often exhibiting longitudinal ridges and lacking the luster of the nails of a younger person. The condition of onychogryphosis, a huge claw-like toe nail, probably the result of neglect, is associated with the elderly.

None of these changes is of serious importance except in that they predispose the altered skin to injuries, infections, and degenerative changes. The margin between ordinary senescent changes and the pathologic is so narrow as to be unrecognizable. It is inevitable, at least in the light of our present ability to control such matters, that the skin will undergo involutional change and even pathologic change, for "it cannot be otherwise than that like barnacles accumulated through the calms and storms of the voyage, the aged must bear cicatrices" of the sun, and wind, and cold, and the thousand and-one irritants to which the skin is repeatedly exposed. Give the skin sensible care and much may be done to prevent the development of serious pathologic conditions.

CARE OF THE SKIN

Bathing. The senile skin should receive the same gentle consideration that is given the skin of an infant. An old skin does not take kindly to too frequent

of one or two cupfuls of a starch product, such as Linat, which is readily soluble in water and does not require preliminary boiling. Borax and sodium bicarbonate, when added to the bath, seem to soften the water and are frequently added to hard water for that purpose, but their soothing effect is deceptive, for they are alkalies and in time have a drying effect on the skin. The addition of other glutinous substances such as bran or almond meal in adequate quantities may also be used to make the water less harsh.

Excessive bathing damages the skin temporarily by thinning the epidermis, but more seriously by removing the sebaceous secretions necessary to the preservation of the skin. In old persons there is usually a diminution of the sebaceous secretions, and in older persons

than to take too frequent tub baths. Baths of extreme temperatures such as the cold bath or the Turkish bath, should be avoided. The ideal bath is a warm one and should not be indulged in for a longer period than fifteen or twenty minutes (See p 133.)

After the bath it is well to anoint the skin with an *oily lotion* or *cream* to supplement the natural fats that the old skin is being deprived of because of

lubricants and most reputable cosmetic manufacturers now produce excellent preparations for the purpose. There is always the likelihood that a hypersensitive skin may be irritated by one of these products, however, even though it may be used safely on most skins. It is well to caution persons with old skin to avoid irritating it by dry air or strong cold winds and rough underwear and particularly, to guard against chafing.

Role of Vitamins. The newer knowledge of the effects of vitamins on the skin suggests that they may be helpful in preventing certain changes peculiar to old skins. There is some evidence, for instance, to indicate a relationship between deficiency of vitamin A and the development of dry skin and scaly eruptions. Perhaps the administration of vitamin A would help retard the development of scaly keratoses and the dry scaly senile skin. The indication for vitamin B complex lies in the fact that in many elderly persons the diet is deficient because of poor teeth or dentures or digestive disturbances and it may be, as is so often claimed, that low grade vitamin B deficiencies are not uncommon among the elderly.

These measures, however helpful, may retard, but not prevent, the development of involutional changes. Unnecessary skin discomforts in the aged may be avoided with these simple rational measures and perhaps even the actual development of pathologic changes may be postponed.

SKIN DISEASES IN OLD AGE

Some of the most troublesome complaints of old age concern the skin. Senile pruritus, for example, can be most distressing. Although the aged skin is exempt from very few diseases, only those of a degenerative character, as keratoses and epitheliomata, are ordinarily associated with senescence. In such diseases as erysipelas and herpes zoster, the factor of old age modifies the usual course, while others, as the lymphoblastomata, although they occur in younger persons, are more prevalent in old age.

In the following paragraphs consideration is given to skin diseases which are *most likely* to affect the aged, with emphasis on the more serious ones.

SENILE KERATOSES

Senile keratoses are among the more important cutaneous lesions in old age because of the frequency with which they undergo malignant change. Any individual after the age of sixty is subject to these degenerative processes, but certain skins appear to be more susceptible and are affected earlier. Such a skin occurs often as a family trait. It is characterized as ruddy and is marked by telangiectasia, dryness, scaling, atrophy, and tiny areas of depigmentation. The predilection of senile keratoses to appear on exposed parts of the face, dorsal surfaces of the hands, and particularly on the bridge of the nose and the temples, suggests that the sun, wind, and other elements may play a causative role in their production. Those persons who have led an out of door life are more disposed to the affection.

Symptoms. Senile keratoses may arise *de novo*, or, more usually, develop upon senile freckles, circumscribed brown pigmented spots in the skin. The lesions vary in size from a split pea to a small fingernail and are covered with closely adherent small thick greasy scales, grayish or yellowish brown in color, which, if removed, produce bleeding. The lesion becomes slightly elevated, roughened, irregular in outline, but remains circumscribed. Although slow to develop, it is persistent. Sooner or later it takes on a verrucous character, with an inflamed base and a red border surrounded by dilated capillaries. This is the beginning of degeneration into epithelioma (Fig. 174). Senile keratoses do not disappear spontaneously.

Treatment. The treatment of senile keratosis depends upon the stage of development of the lesion. Often the lesions can be made to disappear by the

incidence of epithelioma would be reduced considerably if they were to be eliminated and, when possible, prevented

Pathology. Epitheliomata are classified into three types basal cell, squamous cell and mixed (transitional, or basal-squamous-cell cancer) the distinction being based on histologic grounds according to the genesis of the growth On clinical grounds alone one cannot be absolutely certain of the nature of the lesion The squamous-cell growths, although encountered less often, transcend the basal cell types in seriousness because of their tendency to metastasize Due to their rapid development, they are more likely to be ulcerated, vegetative, or fungoid In mucous membranes they may arise as primary growths, but on the skin they arise almost invariably from precancerous lesions Conversely, the basal-cell cancers seldom metastasize, they develop slowly and are thought never to arise as primary growths of mucous membranes The latter, much the more common of the two types, is usually encountered on the face, but not at mucocutaneous junctions

A *basal cell epithelioma* begins either as a small superficial flat thickening or plaque of induration in the skin or as a small firm superficial nodule This lesion has a somewhat translucent waxy appearance and its development is slow, sometimes taking years to reach the size of a split pea It may be shiny, pinkish, yellowish, or even pearly in appearance due to the stretching of the skin over the nodule Gradually the lesion becomes depressed in the center and new nodules make their appearance on the border, perhaps with telangiectases coursing over them The growth continues to spread laterally and then a small crust is seen to cover a tiny central ulcer If the crust is detached bleeding follows As it progresses the ulcer becomes larger and deeper and is surrounded by a characteristic beaded border made up of small pearly nodules traversed by telangiectases Occasionally small growths do undergo spontaneous healing, often, however, this healing is only superficial and actually they continue to develop deeply Basal-cell epitheliomata ordinarily do not give rise to subjective symptoms and, because of their slow evolution and absence of metastasis, they are often considered to be relatively benign, but they are cancerous nevertheless and the danger lies in their tendency to spread deeply to involve underlying important structures such as blood vessels and cartilage

Squamous cell, or prickle cell, cancer may first manifest itself either as a small warty growth raised slightly above the level of the skin, or as a deep-seated nodule, or again as a flat, indurated, rather well demarcated lesion It is painless Clinically it resembles the basal-cell type, but inexplicably the squamous cell cancer has about it a sense of foreboding It grows rather rapidly, is highly vascular, and usually it is surrounded by a slight erythematous halo In a period of but a few months a lesion, which may grow in all directions, becomes infiltrated and ulcerated, and the borders of the ulcer become harder, everted, and undermined, but the characteristic pearly nodules can still be recognized in the border This type of cancer attacks particularly the mucous membranes, the mucocutaneous junctions, the extremities and the genitalia Epitheliomata that develop upon precancerous dermatoses particularly keratoses, scars, or patches of x-ray dermatitis, are usually squamous-cell epitheliomata

The *mixed transitional or basal squamous cell cancers* clinically resemble basal-cell cancers, but suddenly, after a long quiescent period, assume greater

activity Histologic examination will reveal that they are the transitional type, probably metamorphosed from a basal-cell growth into the mixed type or even frank squamous-cell cancer They, too, tend to metastasize

Clinical variants of ordinary epitheliomata occur not infrequently and some assume such characteristic clinical pictures as to warrant distinctive designations for them

Morphea-like epithelioma is a rare type of basal-cell epithelioma which has a superficial resemblance to a small patch of circumscribed scleroderma or morphea It occurs as a rounded, flat, infiltrated patch that in color is a mixture of yellow, white, and pink so that it has a marbled appearance Telangiectatic vessels are present at the periphery and on the surface The surface may be depressed and in time it becomes ulcerated

Bowen's "precancerous" dermatosis is a slow growing epithelioma Beginning as a firm, pale red papule, it soon becomes covered by a scale or crust The papule enlarges to form a lenticular, coin-sized, or discoid lesion, either discretely in plaques or as a confluent mass Beneath the crust the surface is usually red and granular The lesion spreads by peripheral extension, often assuming a polycyclic outline to give it somewhat the appearance of a lesion of tertiary syphilis of the skin Often multiple lesions occur and they have been known to give rise to metastatic growths Surgical excision of these lesions effects better results than does radiation

Paget's disease, which usually attacks the nipple and areola, occasionally involves other areas of the skin in so-called extramammary Paget's disease It is a carcinoma with symptoms of eczematous inflammation Usually there is only one lesion, which has a peculiar red, glazed, moist, eczematous-looking surface When present on the nipple immediate radical removal of the breast is necessary for the deeper intra-epidermic milk ducts will already have been involved with carcinoma For the extramammary types thorough destruction of the lesion will suffice

Multiple benign erythematous epitheliomata are rare basal-cell growths sometimes found on the trunk of old people These lesions are red, slightly thickened areas surrounded by narrow, thread-like borders composed of tiny pearly nodules They look much like lesions of psoriasis The course of the growth is very slow

Pigmented basal cell epithelioma, cystic epithelioma, and lipoepithelioma are other variants with clinical characteristics of a kind to suggest their descriptive names

The term *carcinoma of the skin*, as distinguished from epithelioma, is given to lesions which result from carcinoma of deeper tissues Most often carcinoma of the skin is secondary to carcinoma of the breast and in rare cases the spread of lesions is so extensive as to involve the front and back of the chest with a hard, leathery, boardlike carcinoma termed "cancer en cuirasse"

Diagnosis. The diagnosis of an epithelioma rarely presents any difficulty In the elderly, one must regard with suspicion any new lesion, especially on the head, neck, or hands, which persists, which continues to grow and to bleed from slight trauma, which becomes hardened, indurated, or ulcerated, particularly if it is associated with moles, keratoses, warts or, in fact, with circumscribed lesions of any sort Udo Wile has stressed the "marked tendency to malignant epithelial degeneration in all conditions in which there is a break

in the continuity of normal epithelium." The most characteristic clinical features of an epithelioma are the waxy, hard, pearly border, the nodular character of the base and the free bleeding of ulcerated lesions.

Differential Diagnosis. There are several conditions which simulate epithelioma and must be differentiated from it.

A *syphilitic chancre* resembles epithelioma, particularly if it occurs on the lip or finger, but it can readily be distinguished by the huge regional adenopathy that accompanies a chancre. A darkfield examination will reveal spirochetes if the lesion is seen early and provided it has not received topical treatment with antiseptics, later a serologic test of the blood will give a positive reaction. Unlike epithelioma, the syphilitic chancre tends to regress in a few weeks.

The *gummatous syphiloderm*, a manifestation of tertiary syphilis, consists of rounded firm tumors which eventually soften in the center and break down to form an ulcer of punched out character with a polycyclic outline. This lesion responds quickly to antisiphilitic treatment.

Lupus vulgaris begins usually in childhood. Its lesions are soft, brown, apple-jelly nodules with a microscopic picture that is typical for tuberculosis.

Occasionally a large solitary lesion of *molluscum contagiosum* is mistaken for epithelioma because both have a similar appearance. A typical mol-

agnosis.

Treatment. The treatment of an epithelioma comprises destruction of the lesion by means of excision, electrosurgical measures, irradiation, or a combination of these. A set routine for treatment for all cases cannot be followed, since the choice of method depends upon the location of the epithelioma, its soil, previous treatment, the presence of apparent glandular involvement, and other factors. The greater number of lesions seen in dermatologic practice are small superficial basal-cell lesions which can readily be treated by many different methods and technics.

A *treatment technic* first suggested by Sherwell gives excellent results. The base of the lesion is infiltrated with a few drops of novocain and then curetted with a dull instrument to break down the friable epitheliomatous tissue. This has such a characteristic appearance as to be of diagnostic aid. The friable material and a piece of the "healthy" border are scraped out and sent to a laboratory for microscopic examination. The base and sides are then thoroughly cauterized with either zinc chloride or acid nitrate of mercury. This serves a two fold purpose, it provides chemical destruction of any remaining epitheliomatous tissue and it produces a violent inflammatory reaction which destroys isolated cells. After a few moments, when the patient complains of a burning sensation, the excess of caustic is washed out with water and the area, with surrounding zone of $\frac{1}{4}$ inch of normal skin, is treated by radiation. In about a week a slough begins to separate (care should be taken to prevent the accumulation of pus) and in the course of some six to eight weeks the area heals over completely with a smooth healthy scar which should be examined periodically thereafter for several years.

This method, which combines surgery, caustics, and radiation, offers a wide margin of safety and gives excellent results. It is recommended because it is an office procedure, thus avoiding the need for hospitalization and caus-

ing but a minimum of inconvenience to the patient. It cannot be applied, however, to all epitheliomata for, when lymph nodes are obviously involved from metastasis, treatment must be with a combination of surgery and radiation. It would not be suitable for lesions involving the canthus or for a deeply infiltrated lesion of the lip. For tiny, pea sized lesions on the skin, electrosurgery alone should suffice, or radiation. Perhaps the best single method of treatment for epithelioma is with radiation, the best course, however, is not to follow a single method of treatment for all cases, but to vary the treatment according to indications.

SEBORRHOEIC KERATOSES

Seborrheic keratoses, or seborrheic "warts," are not true warts, strictly speaking. They occur chiefly in those elderly people who have an oily skin and more often in women than in men. At first they are flattened and



Fig. 176 Seborrheic keratosis

yellowish in color, but soon become papillomatous and colored with various shades of brown and, occasionally, black. They increase in size slowly, are usually oval or rounded, as a five-cent piece, and they may attain a thickness of one eighth to one-quarter inch. They usually occur on the temple or cheeks,

LEUKOPLAKIA

Symptoms Leukoplakia is characterized by the gradual development on the mucous membranes of persistent whitish plaques of variable size and

or they may become progressively more indurated and thickened with a rough irregular surface in which fissures may form. As a rule, the affection is limited to a superficial patch or two on the buccal mucous membrane just within the commissures of the mouth. Leukoplakia may involve the tongue or vulva, in fact any mucous membrane, and the involvement may be very extensive. Aside from very slight sensitiveness to heat and cold, the lesions are without subjective symptoms.

Etiology. Leukoplakia very frequently degenerates into squamous-cell cancer, particularly if the patches are subject to repeated irritations. Not all of the factors in their production are known, those that are usually incriminated are smoking, ill-fitting or rough dentures, poor oral hygiene, syphilis and, in rare cases, electrogalvanism due to dissimilar metals in dentures in the mouth. The importance of the role of syphilis has long been questioned by many observers. Nevertheless the safer viewpoint in treating leukoplakia is to "Look back toward syphilis, look ahead to cancer" (Stokes).

Differential Diagnosis. White patches in the mouth caused by lichen planus, monilia infection, or lupus erythematosus are sometimes confused with leukoplakia. *Lichen planus* assumes definite patterns in patches with lesions that are either angular, linear, annular, or fenestrated. Usually there is involvement also of the skin with purplish, flat topped papules. Surface scrapings of the white patches due to *monilia* will readily distinguish it from ordinary leukoplakia, and the patches of *lupus erythematosus* are usually inflammatory, with visible dilated capillaries and red halos around the lesions. They, too, are usually associated with cutaneous lesions.

Treatment. The first step in the treatment of a case of leukoplakia is to eliminate all sources of irritation—tobacco, alcohol, hot liquids, spicy foods, and rough edges on teeth or dentures. These measures, combined with the use of a bland mouth wash, are often sufficient to cause involution of a superficial patch, but indurated patches must be destroyed and preferably by electrosurgical methods, particularly the actual cautery. Radium is preferred by many physicians but, whatever the method, the destruction must be thorough. Anything less than total destruction, as is produced by chemical caustics, is dangerous. It is good practice to examine the patient periodically for some time afterwards. The administration of vitamin A has been credited with effecting involution of superficial patches of leukoplakia.

KRAUROSIS VULVAE

Kraurosis vulvae, a condition characterized by atrophy of the skin and mucous membranes of the vulva, is another of the more serious diseases affecting the aged because it is a precursor of cancer. It is more a concern of gynecologists than dermatologists and is fully considered in books devoted to gynecology.

Treatment. For early cases there have been some rather encouraging results reported recently from the use of hormone therapy and vitamin A. Indeed, hormone therapy is reported to have given some promise in the treatment of leukoplakia, too, whether located in the mouth or in the genitalia.

CUTANEOUS HORNS

Cutaneous horns are rare, but when they occur in the elderly they are serious because they nearly always arise from an epitheliomatous base. They

are masses of hornified epithelium which assume shapes resembling horns. Ordinarily they are about $\frac{1}{4}$ inch in length, of dark gray or brown color, straight, curved, or twisted and with a rough surface. They are usually narrow with a diameter of about $\frac{1}{8}$ inch, but extreme examples both in width and length have been noted. Ordinarily there is but a single lesion usually on the face or scalp but, as with epitheliomata, horns may occur on any part of the body. They usually develop slowly. They spring not only from epitheliomata, but from senile keratoses and occasionally from warts, scars or small sebaceous cysts.

Treatment. They require destruction either by excision or cautery and the base, if epitheliomatous, must be treated by irradiation.



Fig. 177 Cutaneous horn (Patient of Dr. Edward Oliver)

SENILE PRURITUS

Senile pruritus is probably the most annoying condition to plague the aged. It occurs commonly and is usually intractable to treatment. The term

pruritus

Pruritus may be caused in innumerable ways, but it can usually be traced to one of the diseases in the following groups

- 1 Parasitic causes, particularly scabies and pediculosis
- 2 Constitutional diseases diabetes mellitus, nephritis, hepatic disease, endocrine dysfunction, tabes
- 3 Blood dyscrasia lymphoblastoma, especially Hodgkin's disease
- 4 Toxic erythema and urticarial reactions from drugs, foods, and infections

- 5 Pruritus hiemalis and pruritus aestivalis (winter and summer itch)
- 6 Neuroses functional or organic
- 7 Senile pruritus
- 8 Specific skin diseases such as eczema lichen planus

Symptoms In senile pruritus the *itching* is generalized but seldom involves the entire cutaneous surface at one time. It affects the legs chiefly, but it is often pronounced on the trunk. It affects old men chiefly, men with thin, delicate, soft, pliable skins—a skin younger than its wearer's (Stillians). Other signs of involution such as keratoses, hyperpigmented spots and obvious atrophy need not be present and, despite the extreme annoyance produced by the itching, there is relatively little evidence of scratching aside from the smooth, highly polished fingernails of the confirmed scratcher. The condition is aggravated in cold weather. Many cases are seen in association with prostatic hypertrophy with resultant altered urinary output and blood chemistry findings; in others there is evidence only of arteriosclerosis. The fine response of some cases to endocrine therapy suggests that senile pruritus may indeed be a result of "the major involution."

Treatment Soothing applications should be used to alleviate the itching. Preparations such as unguentum aqua rosae for dry skins, or calamine liniment or tragacanth lotion are usually helpful.

1 Boric acid	10%.
Ung. aqua rosae	q s a d
2 Calamine	
Zinc oxide aa	12%.
Lime water	
Olive oil aa	q s a d
3 Tragacanth	5%
Glycerin	2%
Water	q s a d

To these may be added a cooling agent, as menthol 0.25 per cent, or an anti-pruritic agent as phenol 0.5 per cent or liquor carbonis detergens 5 per cent. The best of the internal measures is bromides given intravenously and, more recently, endocrines. Sodium or strontium bromide, 15 grains to the ampule given intravenously twice or three times a week, often effects the greatest relief. There may be some connection in connection.

much to be desired. The introduction of *endocrine therapy* for this condition, however, offers some new hope. Reports indicate that gratifying results may be obtained by the use of 5 mg. of testosterone propionate injected subcutaneously every five days for men, and for subcutaneously every after a few injections.

are encouraging enough to merit attention. If one adds to this the newer knowledge of the effects of vitamin A upon the skin, the future may be a happier one for the old with senile pruritus.

ACAROPHOBIA

Acarophobia is occasionally seen in association with senile pruritus. Fortunately it is rare, for patients with acarophobia are a pitiful lot. They have

the delusion that insects crawl about in their skin and they produce evidence of it in jars filled with lint and dirt of all sorts and epithelial debris which they insist are insects. No amount of reasoning will convince them that it is a delusion unless perhaps expert psychotherapy can do it

PEDICULOSIS CORPORIS

Pediculosis corporis, encountered occasionally among the indigent in dispensary practice, occurs typically in old men, especially in the lodging-house class, where neglect and overcrowding are the rule. Unlike senile pruritus, the amount of scratching in pediculosis corporis is parallel to the intensity of itching, and blood-crusted linear scratch marks are observed on the shoulders, the upper part of the back, arms, thighs, and buttocks—areas that are in contact with the underclothing, the seams of which are the habitat of the organism. Usually there are numerous pyodermic lesions from secondary infection. Where the disease has persisted for any length of time, considerable hyperpigmentation gives the skin the dark brown appearance of "vagabond's disease." The presence of such symptoms should lead to a search in the under-wear seams for pediculi to establish the diagnosis. A cleansing bath, change of clothing, and the application to the skin of unguentum aqua rosae will quickly effect a cure.

SENILE ANGIOMATA

Senile angiomas are the small, soft, red, usually flat lesions which are seen so frequently on the trunk and limbs of the elderly. They are benign and require no treatment, but if they are annoying on the grounds of appearance they can be eradicated easily by freezing with solid carbon dioxide.

ECZEMA DERMATITIS

Cause. Eczema does not respect age. In the aged despite the fact that a dermatitis is a response, in the form of an inflammatory reaction on the part of the superficial epidermal tissues, to the action of an irritant. To produce such a dermatitis there is required *a priori* the factor of hypersensitivity as well as exposure to an irritant, and this factor, hypersensitivity, is less commonly observed in old age. An important reason for the lessened incidence of eczema-dermatitis in the aged is that elderly persons are perhaps less often exposed to irritants. In the production of eczema in the adult the causative irritants are most often of an external nature and are found in chemicals, plants, and their products, biological agents such as bacteria and fungi, and mechanical or physical agents. These irritants, although found anywhere, are more often encountered at work and thus "occupational" eruptions are rare in the old, obviously because so few aged persons are engaged in occupations prone to dispose them to eruptions.

Symptoms and Treatment. As is to be expected, there is a relatively higher incidence of eczema of the legs in the elderly. Venous stasis and the presence of varicose veins are important factors in producing it, but deep-seated inflammations are likely to account for the majority of cases. Infra-malleolar often reveals evidence of phlebitis contiguous with the lower extremity, but with greater involvement of the lower extremities, the eczema may extend up to the knee. In-

inflammation may be observed varying in degree from simple erythema to vesicular eczema, and later thickening, induration, and ulceration may supervene. The ulcers are usually sharply defined, shallow or deep, round or irregular in outline with thickened, firm borders and they have a base covered with a grayish exudate. This ulcer must be differentiated from the polycyclic ulcer of syphilis and from the ulcers of tuberculosis, ecthyma, sickle cell anemia, and simple infected traumatic ulcers. Varicose ulcers are usually surrounded by skin that is eczematous.

Lymphedema with swelling of the leg and thrombophlebitis may result as complications of varicose ulcer. Rest in bed is recommended as the most effective treatment measure. In cases of acute eczema, cool wet packs of aluminum subacetate, 0.5 per cent solution, give immediate relief, later a bland ointment, as zinc paste and petrolatum equal parts, is to be preferred. A supportive bandage should be worn by the patient not confined to bed.

Ulcers are treated similarly, but on occasion do not take kindly to wet dressings or salves. In such cases complete rest in bed and the daily application of 2 per cent aqueous solution of gentian violet will often effect a cure, if not, application of a soft sponge with slight pressure on the ulcer should be tried. In some cases one must rely on the long popular Unna boot or a variation of it (see p. 491). In vigorous persons it may be advisable to eradicate the varicosities as is done in younger persons by preliminary ligation of the saphenous vein, followed by injection of sclerosing agents into the veins of the leg.

Occasionally the ulcer is injured, or the dermatitis is overtreated and the products of exudation are absorbed causing as a result of autosensitization the development of a generalized eczema—the so called *toxic eczematid*. This, too, varies in intensity, but usually fades out in a few days after the application of calamine lotion.

Under the title of *geriatric nutritional eczema* Guy and his associates have reported a group of patients who exhibited edema of the legs (not cardiorenal) associated with a chronic dermatitis. The cutaneous picture is like that in varicose or stasis dermatitis of the legs. The lesions may be erythematous,

simulation, high carbohydrate diet, poor appetite, impaired liver function and general retardation and modification of metabolic processes resulting from involutional tissue changes." A six-point plan of treatment is recommended:

- 1 Adequate mastication to be assured,
- 2 A high protein diet,
- 3 Administration of protein hydrolysates, concentrates, and vitamins,
- 4 Correction of hypochromic anemia,
- 5 Dilute hydrochloric acid with meals,
- 6 Topical care in keeping with the status of the inflammatory process.

Dermatitis from cosmetics makes up a considerable part of the office practice of the dermatologist. It affects chiefly younger women but on occasion is seen in a woman of advanced years, particularly in one who prides herself secretly on looking very little older than her daughter and who employs the necessary measures to attain a youthful appearance. Gray hair is dyed black, straight hair is curled with chemical curling fluid, and so called tissue creams, wrinkle creams, and bleaches are employed in an attempt to stave off the in-

evitable. All such beautifying
are capable of producing
an eruption on the face and
the eyelids.

It
have proved valuable di-

cosmetic irritants

SYPHILIS

Syphilis of the skin in the elderly usually takes a gummatus form, a manifestation of late syphilis. The lesions, when superficial, are designated as *nodular syphiloderm*, the deep subcutaneous lesions are *gummata*.

Symptoms. The lesions occur characteristically in groups that are circinate in arrangement, asymmetrical and without subjective symptoms. There



Fig 178 Nodular syphilis (Patient of Dr. Edward Oliver)

may be a single lesion or several foci may appear in different locations. Cu-

that of arcs of circles with borders almost perpendicular
punched out (Fig 178).

it dries into a dark
a crescentic outline

After healing, a thi... scar remains, on the border of
which new nodules may develop. Gummata occasionally assume variations
such as the psoriasisform gumma or the rupial type—one with a crust resem-
bling an oyster shell. Gummata of the tongue take any of several forms. There

may be crescentic nodules or ulcers, smooth, atrophic, red polycyclic areas, or diffuse nodules which give the appearance of a cobblestone tongue. Serologic tests of the blood are most often, but not invariably, positive at this stage and there may or may not be evidence of syphilitic involvement of other organs. Gummata are not considered contagious lesions. Cutaneous gummata respond readily to treatment.

PERLECHE

Perleche is characterized by the development of scaly, eroded, fissured, or macerated lesions on a red, inflammatory base, situated at the angles of the mouth. The disease always involves both commissures and the lesions cause only slight discomfort. They are observed more often in persons with overhanging upper lips which make the area moist and an excellent culture medium for streptococci or monilia which are the causative organisms.



Fig. 179 Perleche (Patient of Dr. Edward Oliver)

Treatment The presence of perleche in old persons should suggest a deficiency of vitamin B, particularly the riboflavin fraction, and often the lesions will heal with no other treatment than brewers' yeast tablets or riboflavin 1 mg Q I D. The application of 5 per cent ammoniated mercury often hastens resolution (See p. 191).

HERPES ZOSTER

In old people, herpes zoster often leaves distressing persistent pain at the site. The clinical picture is otherwise not dissimilar to that of herpes zoster in younger persons. There are groups of vesicles or papulovesicles on an inflammatory base arranged on the skin in an area supplied by a sensory nerve. The eruption is, therefore, unilateral except in extremely rare instances. In the aged there is a greater tendency to hemorrhagic ulcerated, or even gangrenous lesions and, as already stated, severe postherpetic pain.

Treatment. The eruption is self-limited, usually of about two weeks duration, and requires merely protection with a suspension such as calamine lotion containing 0.5 per cent phenol. The course can sometimes be shortened and the pain lessened by hypodermic injections of posterior pituitary substance, if this is not contraindicated by the presence of cardiovascular disease. Sodium

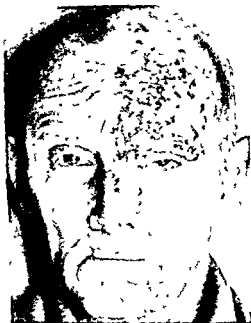


Fig. 180 Herpes zoster

iodide intravenously in doses of 30 grains or autohemotherapy may also be beneficial. When the *postherpetic pain* remains persistent, the treatment is with anodyne drugs, opiates, if necessary, diathermy or radiant heat, and x-ray irradiation over the affected nerve ganglion. Thiamine chloride has been suggested for the treatment of these cases but has not proved successful in my experience.

OTHER SKIN CONDITIONS

Seborrheic dermatitis is also encountered in the aged, as are *erysipelas*, *drug eruptions*, *lichen simplex chronicus*, and *intertriginous eruptions*, particularly in the obese and diabetic. *Purpuric eruptions*, *cutaneous tags*, *fibromata*, *monilial infections*, and the *pyoderms* occur also, even scabies on rare occasions, but their clinical courses are not modified by age. Among the rarer diseases occasionally encountered are the *lymphoblastomata*.



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